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NATIONALGEOGRAPHIC.COM/MAGAZINE

MAY 2005

NATIONAL GEOGRAPHIC

A large tarantula spider with long, hairy legs is the central visual element. One leg is extended downwards, reaching towards a clear glass beaker that sits on a dark surface. The spider's body is partially visible, and its legs are covered in fine hairs. The background is a stark white, which contrasts with the dark spider and the yellow border of the magazine cover.

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THE COVER

A tarantula yields venom used in neural research.

BY CARY WOLINSKY

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CARY WOLINSKY (JOAN CABANISS COLLECTION)

History is full of bad intentions, and this month's cover story on poison is a perfect illustration of that dismal principle. For about as long as humans have been around, poison has been a means to malicious ends. Ancient Athenians accused Spartans of poisoning their wells. Medieval Tartar warriors used plague victims as poison, catapulting them over enemy walls to spread disease. Even today poison seems to be the weapon of choice for some international troublemakers.

Take the news from Ukraine that recently elected President Viktor Yushchenko was the victim of dioxin poisoning. The plot—a real-life crime story complete with a string of strange symptoms, including physical disfigurement—could have come straight out of 15th-century Italy, where poisoning was not only a science but an art.

But while poison can kill, it can also cure. Name your poison, and there's probably a healing side to it. Arsenic has been used to treat leukemia. Mercury was an early treatment for syphilis. It's a matter of dose, and, most of all, it's a matter of intent.

As for our intention: We hope to inform, intrigue, and inspire you—with unexpected tales like that of Leon Fleisher (pages 14-15), the world-famous pianist whose career was reinvigorated thanks to a toxin.

That's the paradox of poison: It occupies the thin line that separates our loftier instincts and achievements from our more sinister side. It's all in the dose.



Lilford's Wall Lizard (*Podarcis lilfordi*)

Size: Length (from tip of nose to tip of tail) 20.4 cm - 22.4 cm **Weight:** 4-12 g

Habitat: Cabrera archipelago and coastal islets of Menorca and Mallorca

Surviving number: Unknown; populations declining



Photographed by Ferran Martí

WILDLIFE AS CANON SEES IT

Meet a plant's best friend. Lilford's wall lizard is more than a little partial to plants—it consumes over 70 plant species. And plants have reason to like the lizard in return. After all, it functions as an efficient seed dispersal system. Research suggests that this particular wall lizard may even act as a pollinator. When it comes to its own progeny, the female lizard hedges its bets by laying several small clutches of one or two eggs. But this

unusual reproductive strategy is not enough to assure the species' survival. Habitat loss and predation—especially by introduced animals—threaten to end a beautiful friendship.

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CHANNEL

MAY 31-JUNE 1, 8 P.M. ET/PT

Mayday! The call for help rings through two days of disaster documentaries filled with heroism, tragedy, and miraculous survival. *Tsunami: Day of Destruction* brings you a firsthand account of what it was like when earthquake-generated waves in the Indian Ocean crashed ashore last December, tearing apart families and villages and inspiring incredible feats of bravery.

In *High-Speed Train Wreck*, see a computer re-creation of a train derailling at 125 mph in Germany. And *Collision on the Runway* captures two jets exploding into a deadly blaze in the Canary Islands.

Heroism plays the lead in *Cruise Ship Rescue*, the story of how all 571 people on board survived the 1991 sinking of the cruise ship *Oceanos* (right) off the South African coast. In *Hijack Rescue*, French commandos save the day when terrorists threaten to blow up a plane over Paris.

Tune in to these programs and more for two days of nonstop action.



MONDAY, MAY 30

9 P.M. ET/PT

Extraterrestrial

News flash 2020: Scientists have found life on two distant planets—and the creatures are like nothing you've seen in the movies. In a spectacular mix of science and animation,



Extraterrestrial enlists leading astronomers to predict what life-forms might exist on other planets. See *Blue Moon* with its dense atmosphere, home to flying sky-whales and three-eyed stalkers. And meet the six-legged mudpod (above) that inhabits the floodplains of Aurelia. Incredible? Watch *Extraterrestrial* and decide for yourself.

Find out what's on and how to get the Channel in your area at nationalgeographic.com/channel. Programming information accurate at press time. Consult local listings.

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SUNDAYS AT 8 P.M. ET/PT

EXPLORER Journey to the front lines of today's most compelling stories. National Geographic Channel's critically acclaimed documentary series *Explorer*, with host Lisa Ling, reports from the frontiers of adventure and exploration, culture and politics, science and natural history. See the world in a new light as upcoming episodes explore why giant locust swarms are relentlessly advancing through Africa, and why natural disasters, from earthquakes to floods, seem to be striking with greater frequency across our planet.

nationalgeographic.com

SIGHTS & SOUNDS Coral Reef Color Explosion What do fish see? How do they use color to attract mates? Dive with photographer Tim Laman to explore their world. **INTERACTIVE QUIZ** Test yourself with facts from this month's articles at nationalgeographic.com/magazine/0505.



CONTEST Win an expedition with one of National Geographic's emerging explorers. How do you realize your potential? Tell us, and you could be the next star of a National Geographic online feature story. **ENTER TODAY** Find official rules for our We See Potential contest at nationalgeographic.com/emerging.

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Selling a home shouldn't be so much work

Trying to sell your home can seem like an up-hill battle against a mountain of paperwork and unexpected obstacles. So why not leave the job to RE/MAX?

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Behind the Scenes

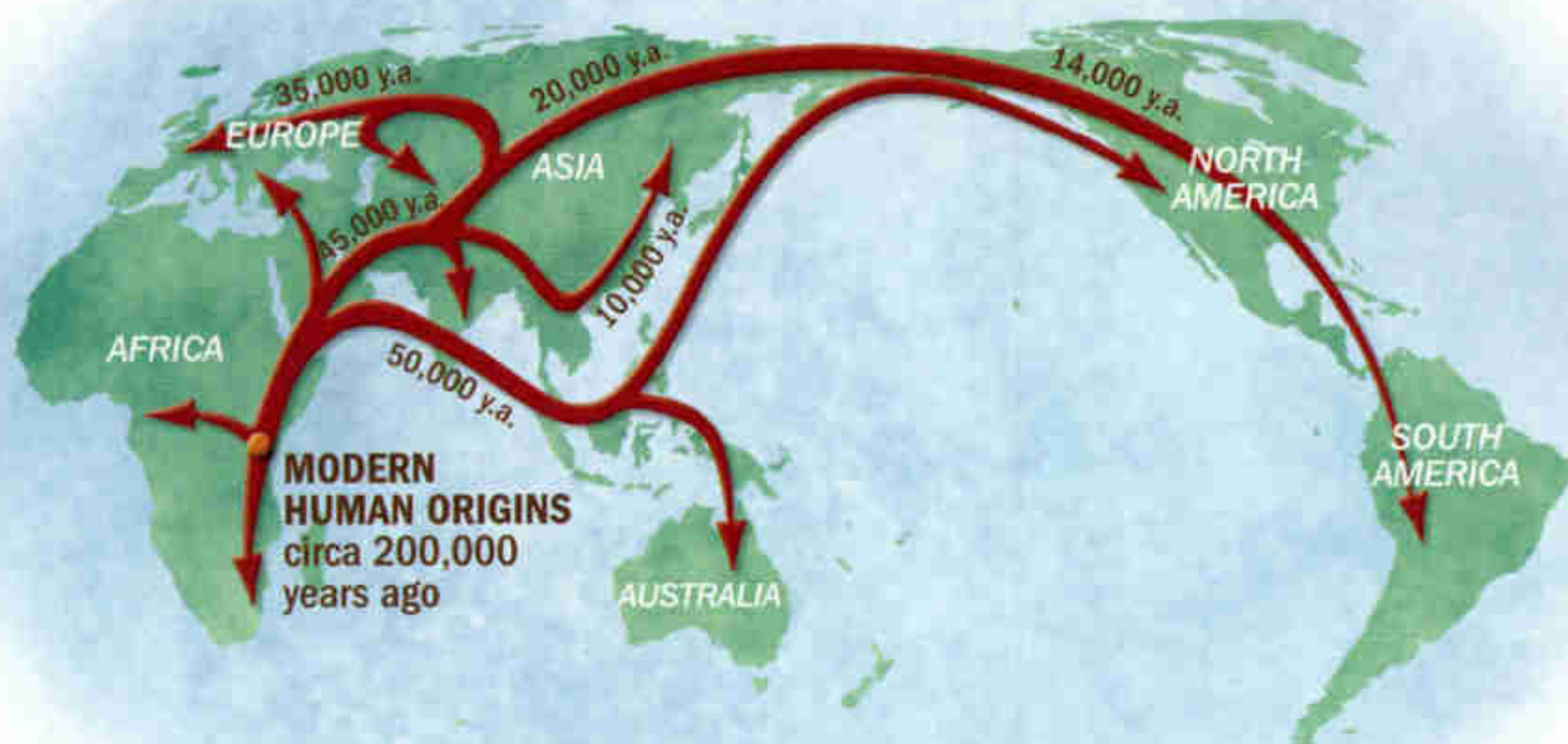
GENOGRAPHIC PROJECT

The Ultimate Family Tree

Most of us can trace family back a few generations, but what if we could look back 60,000 years? Geneticist Spencer Wells hopes to do just that with the Genographic Project, a research partnership of the National Geographic Society and IBM. It's one of our most important—and ambitious—global efforts to date.

"Our DNA is a history book," says Wells, whose genetic work was featured in the October 2004 *GEOGRAPHIC* article, "Who Were the Phoenicians?" Over the course of five years, Wells and his research team—with the financial support of the Waitt Family Foundation—will collect more than 100,000 DNA samples from indigenous populations around the world like Canada's Inuit and Kenya's Masai (right, top two photos). By determining their ancestral migratory routes (below), the project may reveal historical paths that connect people despite their physical and geographical differences.

How might you be connected to a Tibetan woman or an Arizona cowboy (bottom two photos)? Find out—and contribute to the evolving map of human history—by participating in the Genographic Project yourself. Buy a special kit to sample DNA from inside your cheek. Send in the sample to be analyzed and added (anonymously) to the Genographic database. Then go online to watch your personalized ancestral migration map, which will change over time as data arrive from around the world. For more information go to nationalgeographic.com/genographic.



NATIONAL GEOGRAPHIC MAPS

CORAL REEF COLOR (PAGE 86)

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Calendar

APRIL

"Tutankhamun and the Golden Age of the Pharaohs"

exhibit. Ticket sales continue for the June 16 opening at the Los Angeles County Museum of Art, first stop on the exhibit's tour. Call 1-877-TUT-TKTS or go to kingtut.org.

"Survey 2005: In Your Face"

online feature. Participate in a groundbreaking global survey that will help scientists understand how we identify human facial expressions. Go to ngm.com/survey2005.

MAY

10 "Tracing the Journey of Man"

lecture. Spencer Wells of National Geographic's Genographic Project speaks at the Field Museum's James Simpson Theatre in Chicago. Call 312-665-7400 for tickets. On May 18 Wells speaks at the National Geographic Society, Washington, D.C.

12 *The Lost Amazon: The Photographic Journey of Richard Evans Schultes*

Explorer-in-Residence Wade Davis discusses his book about the renowned ethnobotanist at National Geographic, Washington, D.C.

JUNE

13 *Inside the Mafia*

This National Geographic Channel two-night special takes a historical look at the rise of organized crime. Aired June 13 and 14 at 9 p.m. ET/PT.

Calendar dates are accurate at press time; please go to nationalgeographic.com or call 1-800-NGS-LINE (647-5463) for more information.

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THROUGH A PHOTOGRAPHER'S EYE

Visions of Earth

WEST OF GREAT EXUMA ISLAND, BAHAMAS

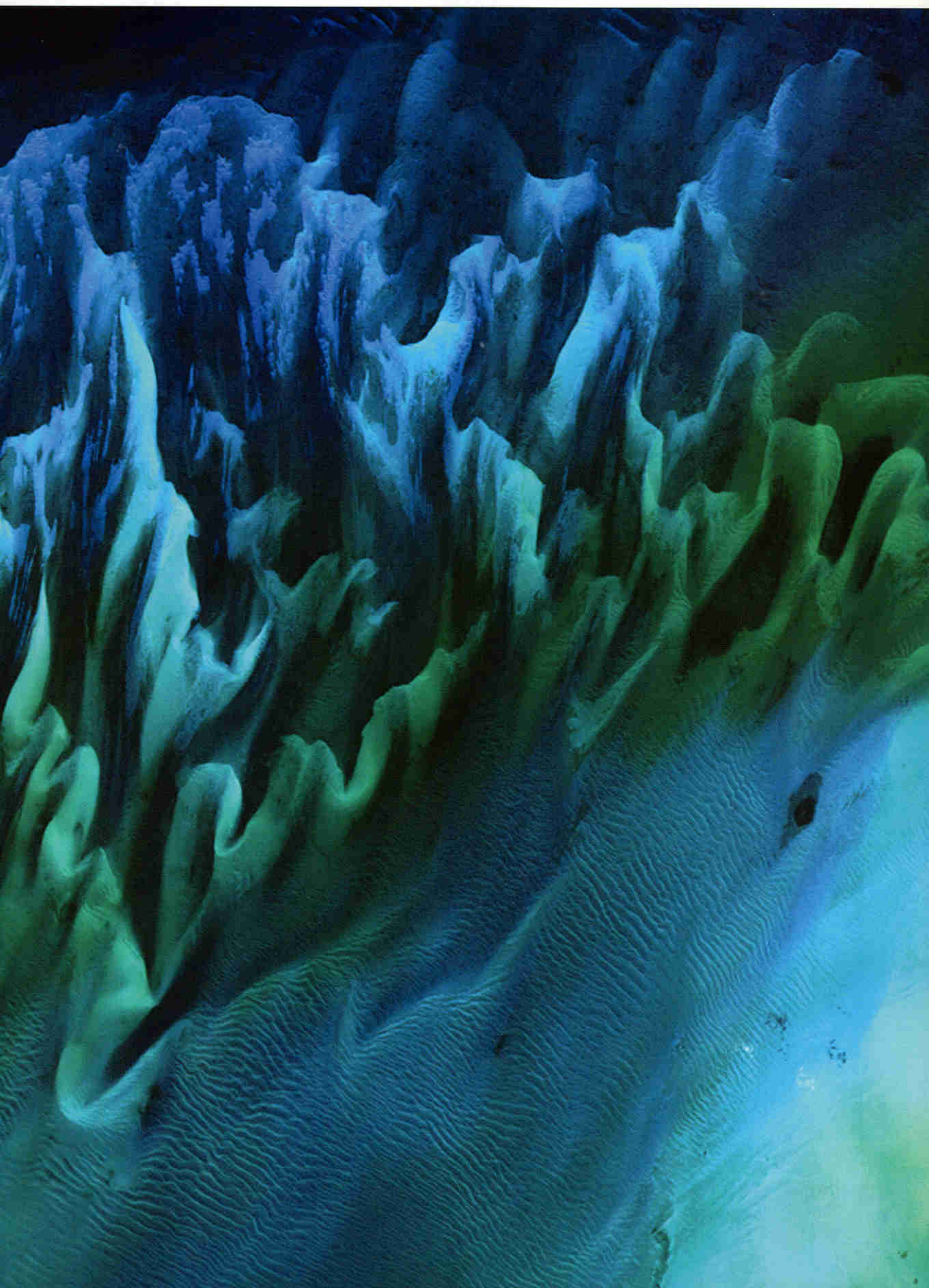
At the tip of a trough called the Tongue of the Ocean, the waters are very clear. And a scan by a sensor aboard the Landsat 7 satellite during a pass over the Bahamas captures their clarity. This 350-square-mile swath is rendered so sharply that even current-carved sands and sea grass some 50 feet below the ocean's surface are visible from space. This is a highly technical image—and yet it keeps some mystery in its depths.

—Serge Andréfouët and Frank Müller-Karger

REMOTE-SENSING SCIENTISTS

► Decorate your desktop with waters off the Bahamas at nationalgeographic.com/magazine/0505.

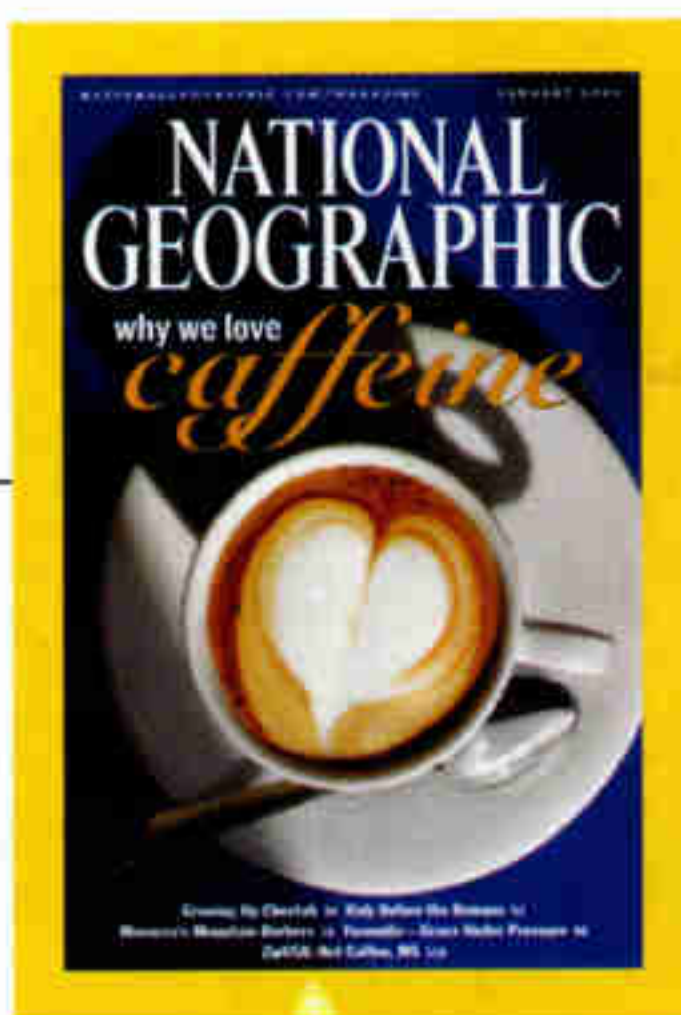
NASA; USGS



Forum

January 2005

This month readers were buzzing about caffeine. Some felt we should have concentrated on coffee farmers in developing countries; others wanted more information on the negative health effects of the stimulant. When it came to the ZipUSA story "Hot Coffee, Mississippi" though, readers praised the writing and photography, giving it one of the warmest responses for any article in the series to date.



Caffeine

I am impressed with and congratulate T. R. Reid for his bio-psycho-socio-economic rendition of caffeine. As a medical doctor practicing consultative and clinical environmental medicine, I am dismayed at the magnitude of coffee use throughout the world. The people I see as patients are tragic victims of environmental toxins of all kinds, including caffeine.

JOHN G. HIPPS
Emporium, Pennsylvania

"What's the Buzz?" is an awesome presentation about caffeine's sources and use in today's world. However, I believe that you have left out an important piece of the picture by not including information on "fair trade" coffee, tea, and cocoa. Few Americans realize that agricultural workers in these businesses usually toil in what can be

described as sweatshops in the fields. Many small coffee farmers in underdeveloped countries receive prices for their coffee that are less than the costs of production, forcing them into a cycle of poverty and debt. Fair trade is a viable solution to this crisis, assuring consumers that the coffee we drink can be purchased under fair conditions.

MARIE O'ROURKE
Cassadaga, New York

I was shocked by your indulgent and biased portrayal of caffeine addiction, which read like a 32-page ad for Starbucks and Red Bull. Your readers have a right to balanced presentations that include serious discussion of a subject as disturbing as substance addiction. What's next, an article on "The Pleasures of Alcohol" or even "The Wonderful High of Heroin"?

KRISTIN A. KUCKELMAN
Dolores, Colorado

I was quite surprised to find no mention that caffeine can cause heartbeat irregularity. I was plagued with this symptom for several weeks. It increased in severity to the point that my husband drove me to the emergency room one morning at

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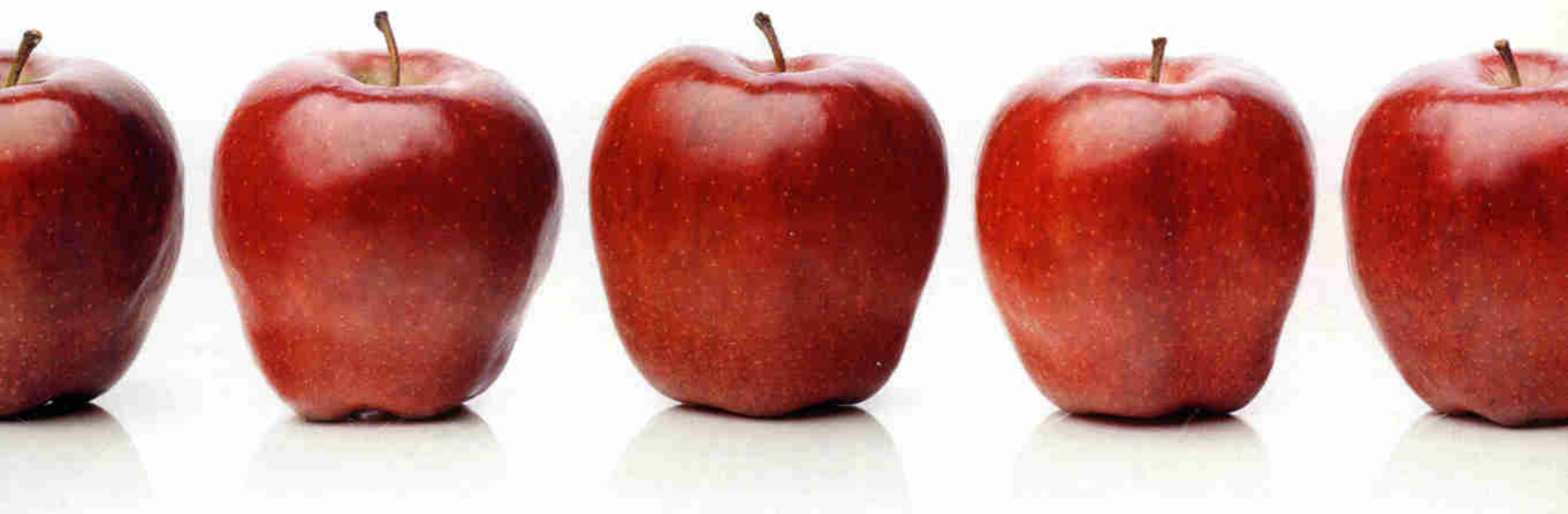
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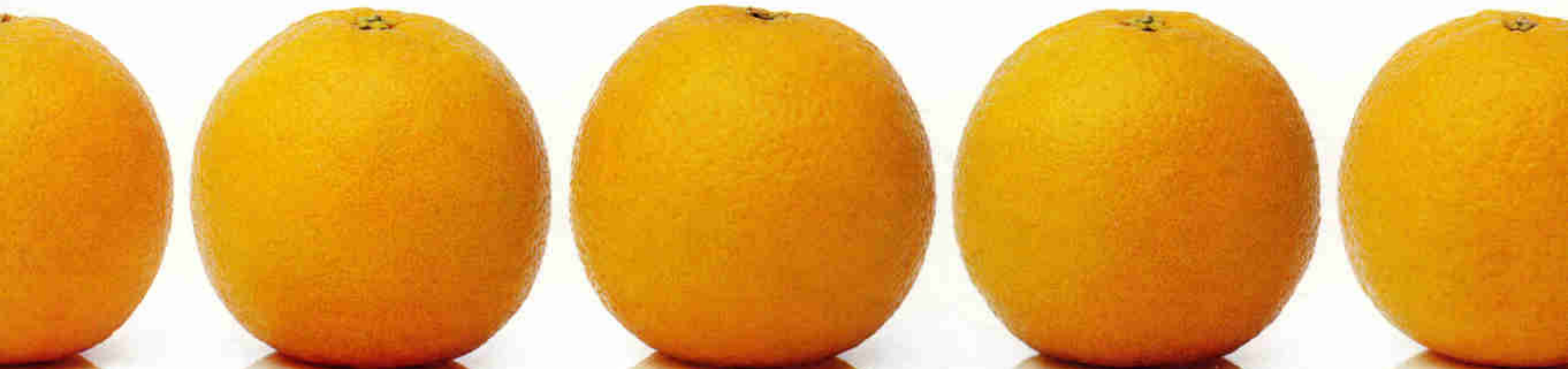
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Generic name: montelukast (mon-te-LOO-kast) sodium

Read this information before you start taking SINGULAIR®. Also, read the leaflet you get each time you refill SINGULAIR, since there may be new information in the leaflet since the last time you saw it. This leaflet does not take the place of talking with your doctor about your medical condition and/or your treatment.

What is SINGULAIR*?

- SINGULAIR is a medicine called a leukotriene receptor antagonist. It works by blocking substances in the body called leukotrienes. Blocking leukotrienes improves asthma and seasonal allergic rhinitis (also known as hay fever). SINGULAIR is not a steroid.

SINGULAIR is prescribed for the treatment of asthma and seasonal allergic rhinitis:

1. Asthma.

SINGULAIR should be used for the long-term management of asthma in adults and children ages 12 months and older.

Do not take SINGULAIR for the immediate relief of an asthma attack. If you get an asthma attack, you should follow the instructions your doctor gave you for treating asthma attacks. (See the end of this leaflet for more information about asthma.)

2. Seasonal Allergic Rhinitis.

SINGULAIR is used to help control the symptoms of seasonal allergic rhinitis (sneezing, stuffy nose, runny nose, itching of the nose) in adults and children ages 2 years and older. (See the end of this leaflet for more information about seasonal allergic rhinitis.)

Who should not take SINGULAIR?

Do not take SINGULAIR if you are allergic to SINGULAIR or any of its ingredients.

The active ingredient in SINGULAIR is montelukast sodium.

See the end of this leaflet for a list of all the ingredients in SINGULAIR.

What should I tell my doctor before I start taking SINGULAIR?

Tell your doctor about:

- **Pregnancy:** If you are pregnant or plan to become pregnant, SINGULAIR may not be right for you.
- **Breast-feeding:** If you are breast-feeding, SINGULAIR may be passed in your milk to your baby. You should consult your doctor before taking SINGULAIR if you are breast-feeding or intend to breast-feed.
- **Medical Problems or Allergies:** Talk about any medical problems or allergies you have now or had in the past.
- **Other Medicines:** Tell your doctor about all the medicines you take, including prescription and non-prescription medicines, and herbal supplements. Some medicines may affect how SINGULAIR works, or SINGULAIR may affect how your other medicines work.

How should I take SINGULAIR?

For adults and children 12 months of age and older with asthma:

- Take SINGULAIR once a day in the evening.
- Take SINGULAIR every day for as long as your doctor prescribes it, even if you have no asthma symptoms.
- You may take SINGULAIR with food or without food.
- If your asthma symptoms get worse, or if you need to increase the use of your inhaled rescue medicine for asthma attacks, call your doctor right away.
- **Do not take SINGULAIR for the immediate relief of an asthma attack.** If you get an asthma attack, you should follow the instructions your doctor gave you for treating asthma attacks.
- Always have your inhaled rescue medicine for asthma attacks with you.
- Do not stop taking or lower the dose of your other asthma medicines unless your doctor tells you to.
- If your doctor has prescribed a medicine for you to use before exercise, keep using that medicine unless your doctor tells you not to.

For adults and children 2 years of age and older with seasonal allergic rhinitis:

- Take SINGULAIR once a day, at about the same time each day.

- Take SINGULAIR every day for as long as your doctor prescribes it.
- You may take SINGULAIR with food or without food.

How should I give SINGULAIR oral granules to my child?

Do not open the packet until ready to use.

SINGULAIR 4-mg oral granules can be given either:

- directly in the mouth;
- OR
- mixed with a spoonful of one of the following soft foods at cold or room temperature: applesauce, mashed carrots, rice, or ice cream. Be sure that the entire dose is mixed with the food and that the child is given the entire spoonful of the mixture right away (within 15 minutes).

IMPORTANT: Never store any oral granule/food mixture for use at a later time. Throw away any unused portion.

Do not put SINGULAIR oral granules in liquid drink. However, your child may drink liquids after swallowing the SINGULAIR oral granules.

What is the daily dose of SINGULAIR for asthma or seasonal allergic rhinitis?

For Asthma (Take in the evening):

- One 10-mg tablet for adults and adolescents 15 years of age and older,
- One 5-mg chewable tablet for children 6 to 14 years of age,
- One 4-mg chewable tablet or one packet of 4-mg oral granules for children 2 to 5 years of age, or
- One packet of 4-mg oral granules for children 12 to 23 months of age.

For Seasonal Allergic Rhinitis (Take at about the same time each day):

- One 10-mg tablet for adults and adolescents 15 years of age and older,
- One 5-mg chewable tablet for children 6 to 14 years of age, or
- One 4-mg chewable tablet or one packet of 4-mg oral granules for children 2 to 5 years of age.

What should I avoid while taking SINGULAIR?

If you have asthma and if your asthma is made worse by aspirin, continue to avoid aspirin or other medicines called non-steroidal anti-inflammatory drugs while taking SINGULAIR.

What are the possible side effects of SINGULAIR?

The side effects of SINGULAIR are usually mild, and generally did not cause patients to stop taking their medicine. The side effects in patients treated with SINGULAIR were similar in type and frequency to side effects in patients who were given a placebo (a pill containing no medicine).

The most common side effects with SINGULAIR include:

- stomach pain
- stomach or intestinal upset
- heartburn
- tiredness
- fever
- stuffy nose
- cough
- flu
- upper respiratory infection
- dizziness
- headache
- rash

Less common side effects that have happened with SINGULAIR include (listed alphabetically): agitation including aggressive behavior, allergic reactions (including swelling of the face, lips, tongue, and/or throat, which may cause trouble breathing or swallowing), hives, and itching, bad/vivid dreams, increased bleeding tendency, bruising, diarrhea, drowsiness, hallucinations (seeing things that are not there), hepatitis, indigestion, inflammation of the pancreas, irritability, joint pain, muscle aches and muscle cramps, nausea, palpitations, pins and needles/ numbness, restlessness, seizures (convulsions or fits), swelling, trouble sleeping, and vomiting.

Rarely, asthmatic patients taking SINGULAIR have

experienced a condition that includes certain symptoms that do not go away or that get worse. These occur usually, but not always, in patients who were taking steroid pills by mouth for asthma and those steroids were being slowly lowered or stopped. Although SINGULAIR has not been shown to cause this condition, **you must tell your doctor right away if you get one or more of these symptoms:**

- a feeling of pins and needles or numbness of arms or legs
- a flu-like illness
- rash
- severe inflammation (pain and swelling) of the sinuses (sinusitis)

These are not all the possible side effects of SINGULAIR. For more information ask your doctor or pharmacist.

Talk to your doctor if you think you have side effects from taking SINGULAIR.

General Information about the safe and effective use of SINGULAIR

Medicines are sometimes prescribed for conditions that are not mentioned in patient information leaflets. Do not use SINGULAIR for a condition for which it was not prescribed. Do not give SINGULAIR to other people even if they have the same symptoms you have. It may harm them. **Keep SINGULAIR and all medicines out of the reach of children.**

Store SINGULAIR at 25°C (77°F). Protect from moisture and light. Store in original package.

This leaflet summarizes information about SINGULAIR. If you would like more information, talk to your doctor. You can ask your pharmacist or doctor for information about SINGULAIR that is written for health professionals.

What are the ingredients in SINGULAIR?

Active ingredient: montelukast sodium

SINGULAIR chewable tablets contain aspartame, a source of phenylalanine.

Phenylketonurics: SINGULAIR 4-mg and 5-mg chewable tablets contain 0.674 and 0.842 mg phenylalanine, respectively.

Inactive ingredients:

- **4-mg oral granules:** mannitol, hydroxypropyl cellulose, and magnesium stearate.
- **4-mg and 5-mg chewable tablets:** mannitol, microcrystalline cellulose, hydroxypropyl cellulose, red ferric oxide, croscarmellose sodium, cherry flavor, aspartame, and magnesium stearate.
- **10-mg tablet:** microcrystalline cellulose, lactose monohydrate, croscarmellose sodium, hydroxypropyl cellulose, magnesium stearate, hydroxypropyl methylcellulose, titanium dioxide, red ferric oxide, yellow ferric oxide, and carnauba wax.

What is asthma?

Asthma is a continuing (chronic) inflammation of the bronchial passageways which are the tubes that carry air from outside the body to the lungs.

Symptoms of asthma include:

- coughing
- wheezing
- chest tightness
- shortness of breath

What is seasonal allergic rhinitis?

- Seasonal allergic rhinitis, also known as hay fever, is an allergic response caused by pollens from trees, grasses and weeds.
- Symptoms of seasonal allergic rhinitis may include:
 - stuffy, runny, and/or itchy nose
 - sneezing

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Yosemite—Grace Under Pressure

Since I live in California, it is a ritual to return to the park every year or so to bask in its grandeur and simple serenity. Yet my most recent trip to the park left me rather concerned. It seems the average visitor believes Yosemite is a natural amusement park.

ESTEFANI MORALES
Antioch, California

Please, write about the beauty of our national parks. Describe the backcountries that most of us can't find the time, money, or strength to enjoy. But don't describe us as rude, uncaring dolts annoyingly obstructing the trails and views you wish to claim exclusively as your own. We, too, want to taste and feel the wonderment as best as our insignificant selves



GALEN ROWELL, MOUNTAIN LIGHT

can, without being ridiculed for doing so.

OTIS HEADLEY
Oneonta, Alabama

The article notes that "beyond the herds of tourists there is another Yosemite—less familiar, perhaps, but charged with a power of its own." It is Hetch Hetchy Valley, the place John Muir called "a grand landscape garden." Unfortunately, in 1913 Congress allowed San Francisco to build a dam and reservoir in the valley. Our organization is spearheading a movement to obtain a

win-win outcome for Hetch Hetchy Valley, the San Francisco Bay Area water and power users, local irrigation districts, and Native Americans whose ancestors lived in the valley.

RON GOOD
*Executive Director
Restore Hetch Hetchy
Sonoma, California*

William Least Heat-Moon calls the Park Service's plan for the park "sensible." What its proponents will tell you sounds good, but they do not tell you how many trees are marked for felling, how much new asphalt is to be laid, and how many new hotel units and employee housing dormitories are planned for the valley floor.

BENJAMIN ZARTMAN
Mariposa, California

4 a.m. The next day my doctor informed me that caffeine can cause heart palpitations. That very day I stopped drinking tea with caffeine and replaced it with decaf tea. It has now been four years since my no-caffeine decision. I still enjoy my hot tea and iced tea, but always decaf.

BETH BURGAMY ZAKRASEK
Cedar Rapids, Iowa

Your article acknowledges that caffeine "boosts" blood pressure but asserts that "this effect is temporary." While it is true that

the blood-pressure-elevating effect of a single cup of coffee or tea is relatively short-lived, the effect of caffeine as ordinarily consumed should not be described as temporary. In reality, average consumers, drinking three to four caffeinated beverages a day, experience elevated blood pressure for most of their waking hours. Consequently, over the course of a lifetime of use, caffeine may contribute directly to increased cardiovascular death and disease. The overwhelming popularity of coffee, tea, and caffeinated soft drinks should not be allowed to obscure their potential health consequences.

JACK E. JAMES
*National University of Ireland, Galway
Galway, Ireland*
JAMES D. LANE
*Duke University Medical Center
Durham, North Carolina*

It was a good article, but there was one thing that bothered me. I noticed that while you printed the name of the photographer who took photos of the latte art, the name of the latte artist was not printed. As a latte artist myself, I realize how much time and effort it takes to perfect the art.

BECKY GUTTERIDGE
Prince George, British Columbia

Luca Mastantuoni made the cappuccino on the cover and the one in the article (pages 32-3). An instructor at the Lavazza Training Center outside Turin, Italy, he teaches baristas to make the perfect espresso or cappuccino.

Among the Berbers

I thoroughly enjoyed Jeffrey Tayler's fine article on Morocco's *mountain Berbers*. The biggest things I felt when I went to

WRITE TO FORUM National Geographic Magazine, PO Box 98199, Washington, DC 20090-8199, or by fax to 202-828-5460, or via the Internet to ngsforum@nationalgeographic.com. Include name, address, and daytime telephone. Letters may be edited for clarity and length.

Morocco to see the High Atlas mountains were guilt, shame, and pity. I felt guilty that I lived in the richest country in the world, and here I was, surrounded by thousands of starving people. When I arrived, I pitied those people. But when I left, I realized it was me who should be pitied. Those people, even though they have nothing, have something the world desperately needs: faith and hope.

EVAN DALE SANTOS
Adelanto, California

Berber life sounds tough for both men and women, but your article seems to denigrate Berber men by saying that women "do the heavy lifting." At the end of the article you note that these men have the privilege of living in shantytowns on the outskirts of Casablanca or Rabat, where they work long hours for a pittance to send home. This doesn't sound particularly easy either.

STEVEN G. VAN VALKENBURG
Westminster, Colorado

Growing Up Cheetah

I was shocked to learn only about 5 percent of cheetah cubs on East Africa's plains ever reach adulthood, and the animals' average life expectancy is merely seven years. I would have expected the world's fastest land mammal to be a champion survivor.

SCOTT MAMMOSER
Eden, New York

Italy Before the Romans

I loved your article on Italy before the Romans. I am the second generation descended from an Italian family to be born in St. Paul, Minnesota. When I was a little girl, my father would make the *malocchio* ritual over my head and heart when I had a

headache. I now perform the ritual on my Irish husband and sons. We all seem to feel better afterward. Thanks to your article I know where this ritual that my Catholic father practiced came from.

VICTORIA MASTRO KANE
St. Paul, Minnesota

Like the sublime
pause between two
symphonic move-
ments, Dan Brooks's
Visions of Earth
photograph seems
to hold time ransom.
It is our Earth at
its most majestic,
inspiring awe and
contemplation.

ZipUSA: Hot Coffee, Mississippi

Thank you for the article about a town in the often neglected state of Mississippi. Peter Gwin's writing, along with the online magazine features and Bob Sacha's fine photography, made me feel like packing my bags to join my fellow Anabaptists in Mississippi.

ALES DVORAK
El Cajon, California

I've always known about Hot Coffee and have visited the area many times in my life, but never did I expect to open up NATIONAL GEOGRAPHIC and see a fantastic article on the heritage and people of the lovely place.

I have always assumed that the people who lived there were Mennonites, but now I know they aren't. I now understand these people a little better.

VICKIE COOPER
Huntsville, Alabama

Visions of Earth

Like the sublime pause between two symphonic movements, Dan Brooks's photograph seems to hold time ransom. It is our Earth at its most majestic, inspiring awe and contemplation. In the wake of the recent Asian quake and the devastation that followed, I cannot but see a more sobering side. The plume of smoke hovering above Semeru's cone seems to remind us of our precarious and fragile existence on this planet by offering us a glimpse of nature's power.

SUBIR CHATTERJEE
Mumbai, India

Geographica: Geo Quiz "Marking the Year"

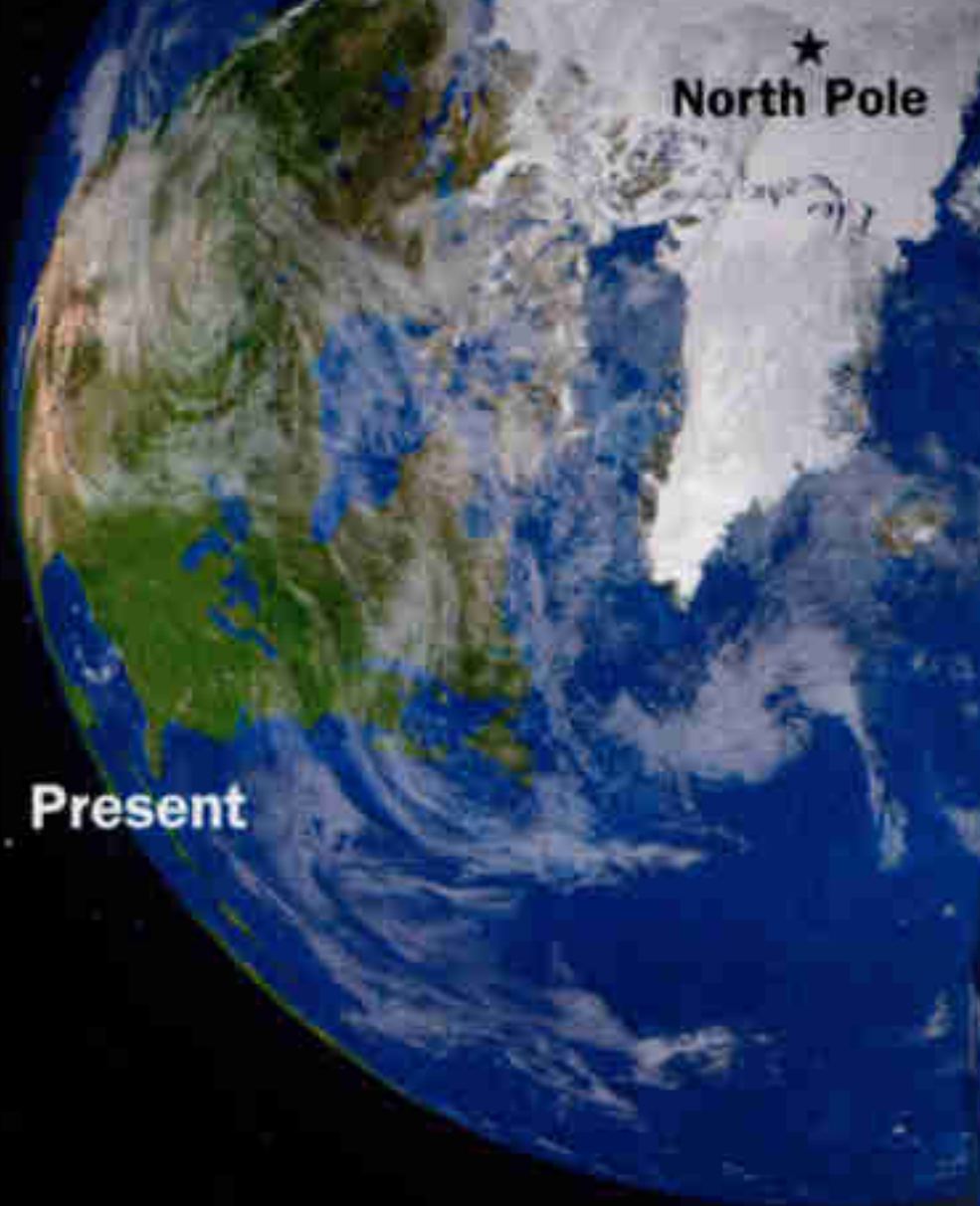
The answer given in your Geo Quiz that Julius Caesar decided the year would begin on January 1 under the Julian calendar is technically correct but is misleading. It conveys the impression that Caesar was the first to select January 1 as the start of the year and that he is the source of our present custom. In fact, the Romans began using January 1 as the start of the civil year in 153 B.C. in their old calendar; so Caesar was simply continuing the custom in his new calendar.

PAUL W. DICKSON, JR.
Aiken, South Carolina

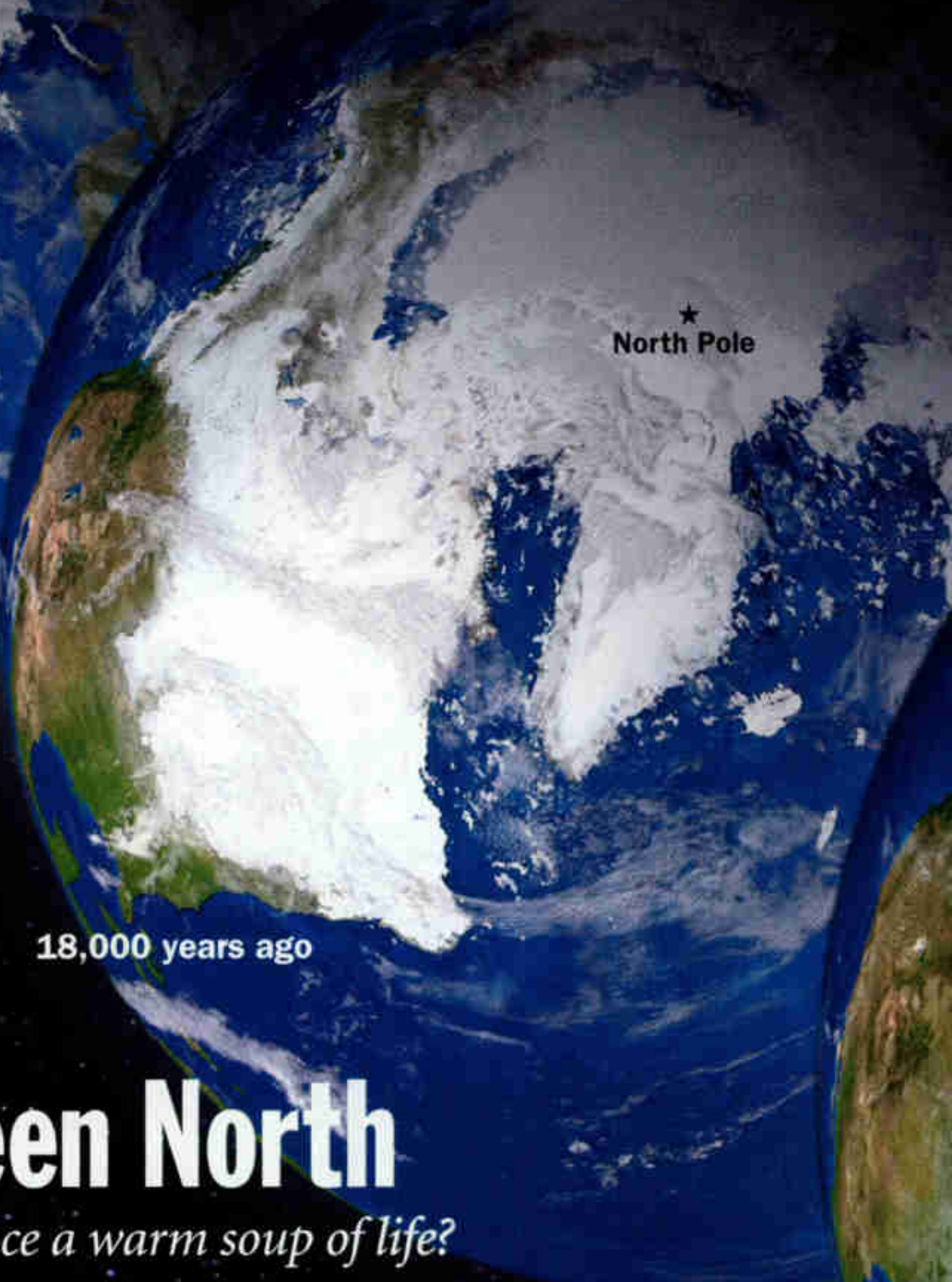
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GEOGR

T H E P E O P L E , P L A C E S , A N D



Present



18,000 years ago

CLIMATE

Great Green North

Was the icy Arctic once a warm soup of life?

Under a microscope they look like greenish brown globs—run-of-the-mill pond scum would look about the same. But these globs are actually clumps of spores from an aquatic fern, and they're revolutionizing our understanding of the Arctic Ocean. Fifty million years ago the now ice-locked northern ocean may have resembled a giant freshwater pond, choked with waterweed and teeming with microscopic life.

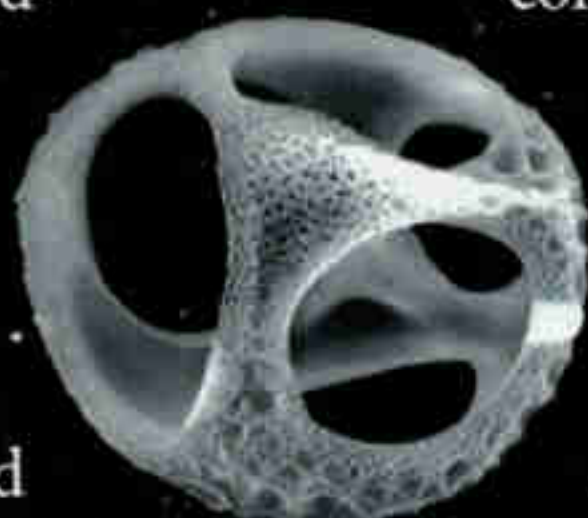
Scientists got their first glimpse of the fern spores aboard an ice-breaker last summer. "I was absolutely shocked," says Ted Moore, a scientist on the Arctic Coring Expedition, an audacious attempt

to bore into the ocean floor. The 15-foot-long cylinders of rock and mud the expedition pulled up may help solve one of Earth's great mysteries: How did the far north, which once enjoyed a balmy climate, become the cold white place it is today?

While other oceans have long been well mapped, the Arctic has remained largely a blank. As recently as 1996 a ship plowing through pack ice near

the North Pole found that existing charts had misplaced a giant underwater ridge by 120 miles, and an ocean-floor abyss, shown as featureless, was actually a welter of mountains and canyons.

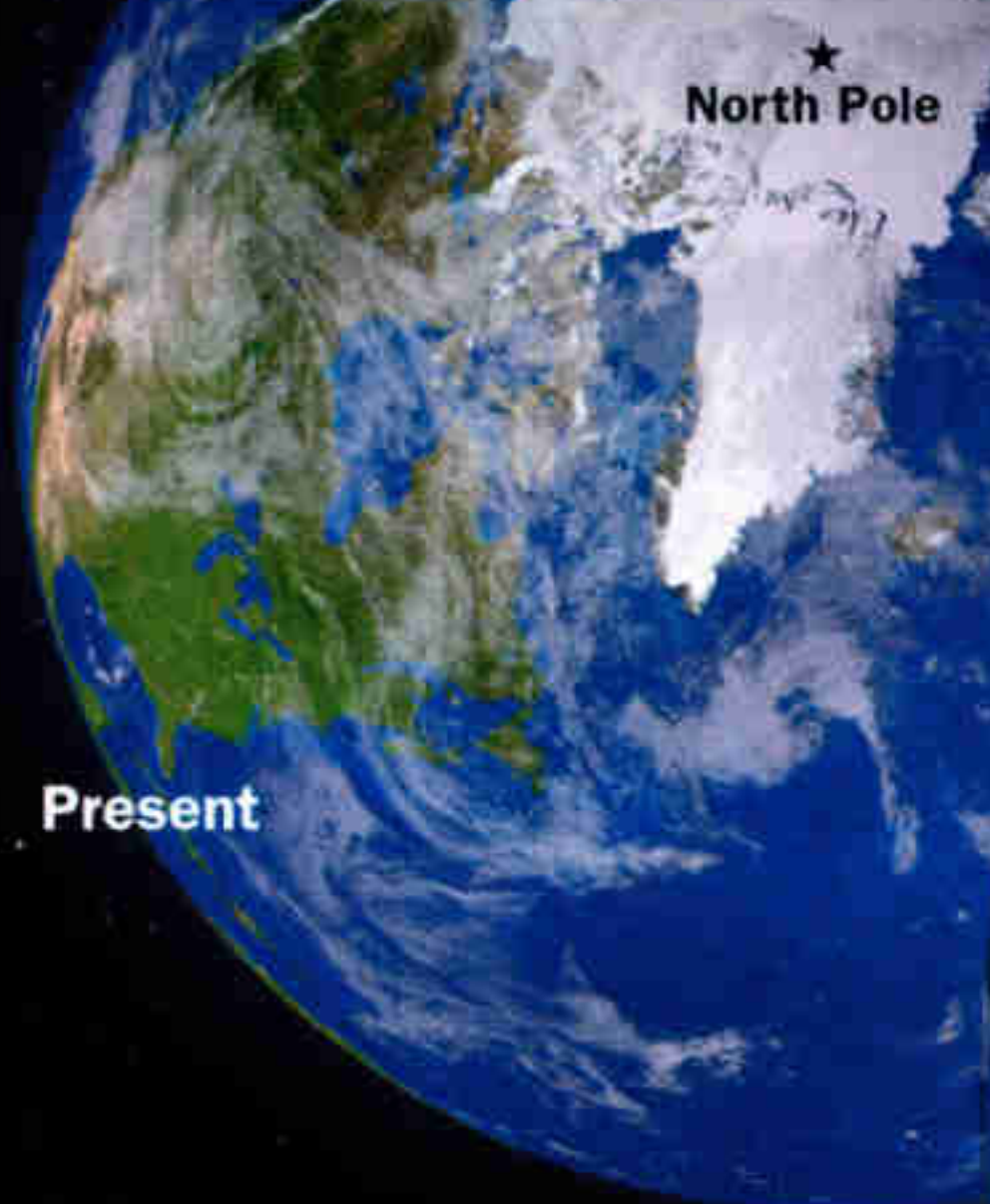
More mysterious than the



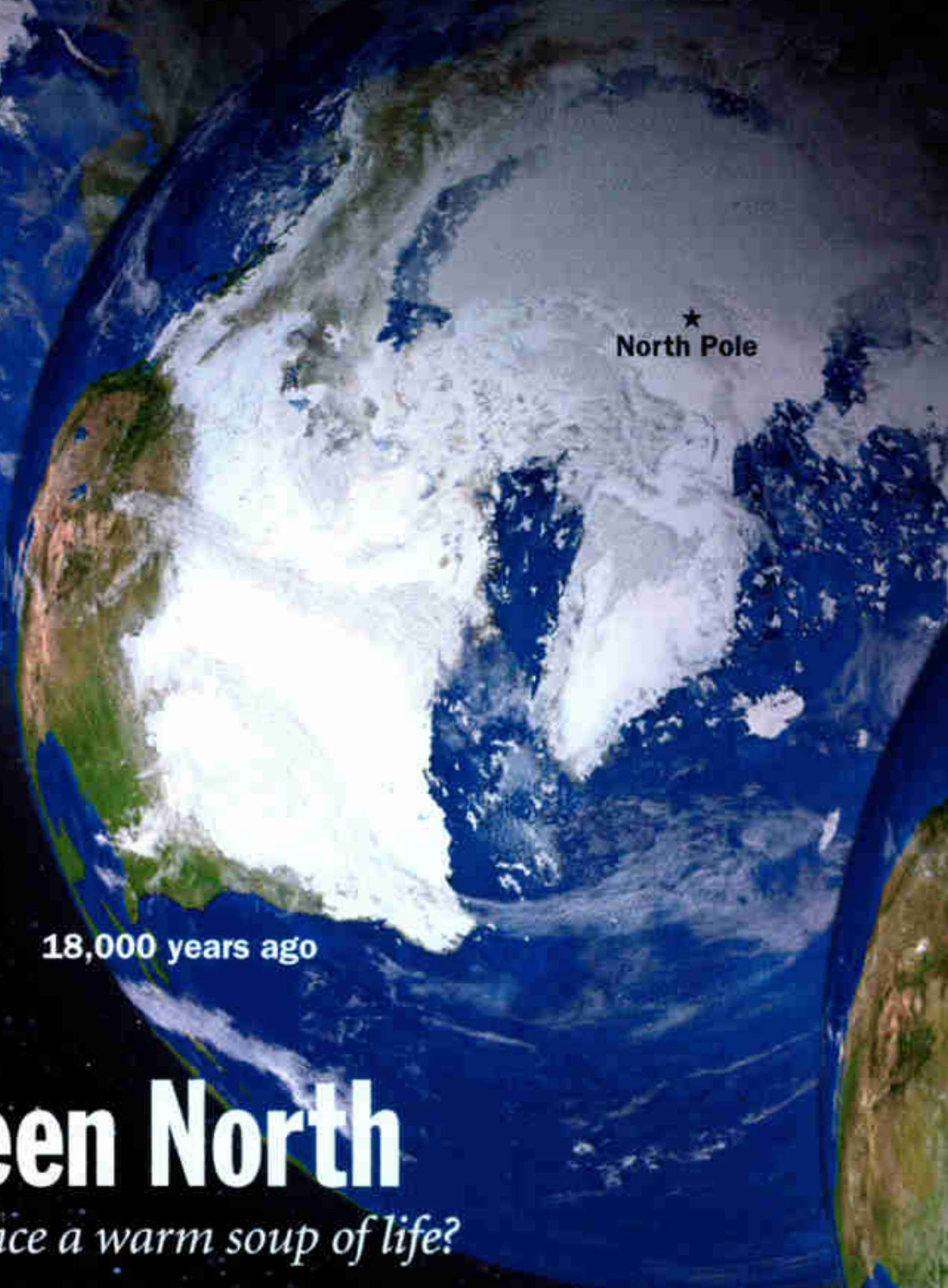
14 million years ago

GEOGR

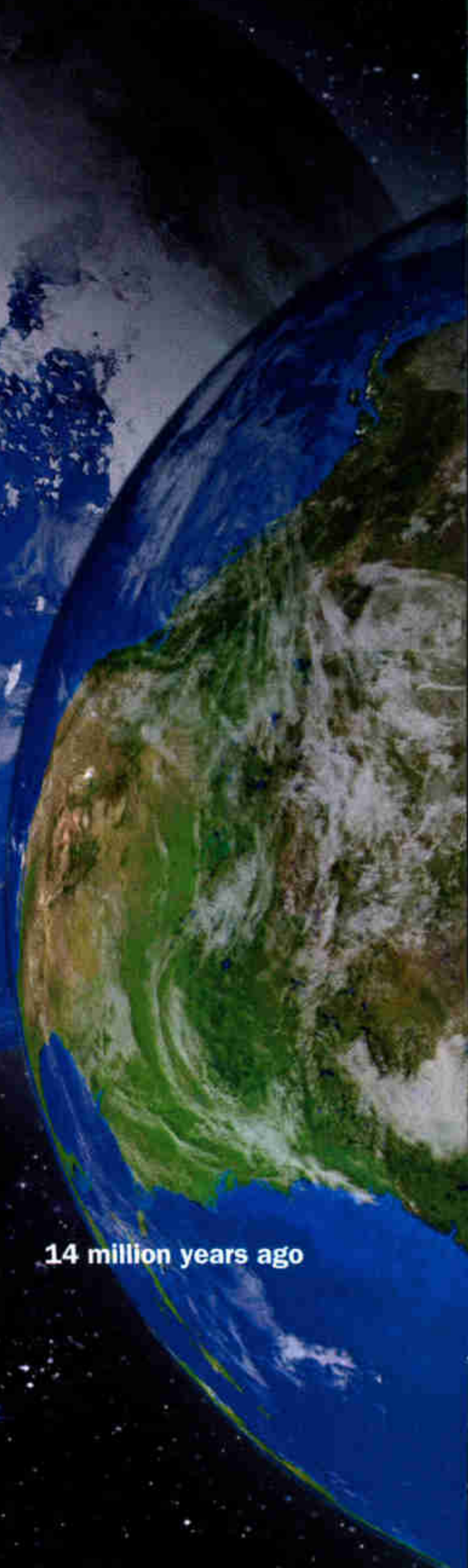
T H E P E O P L E , P L A C E S , A N D



Present



18,000 years ago



14 million years ago

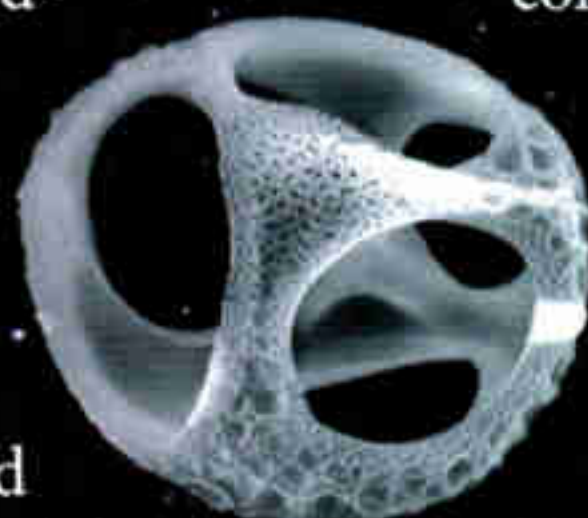
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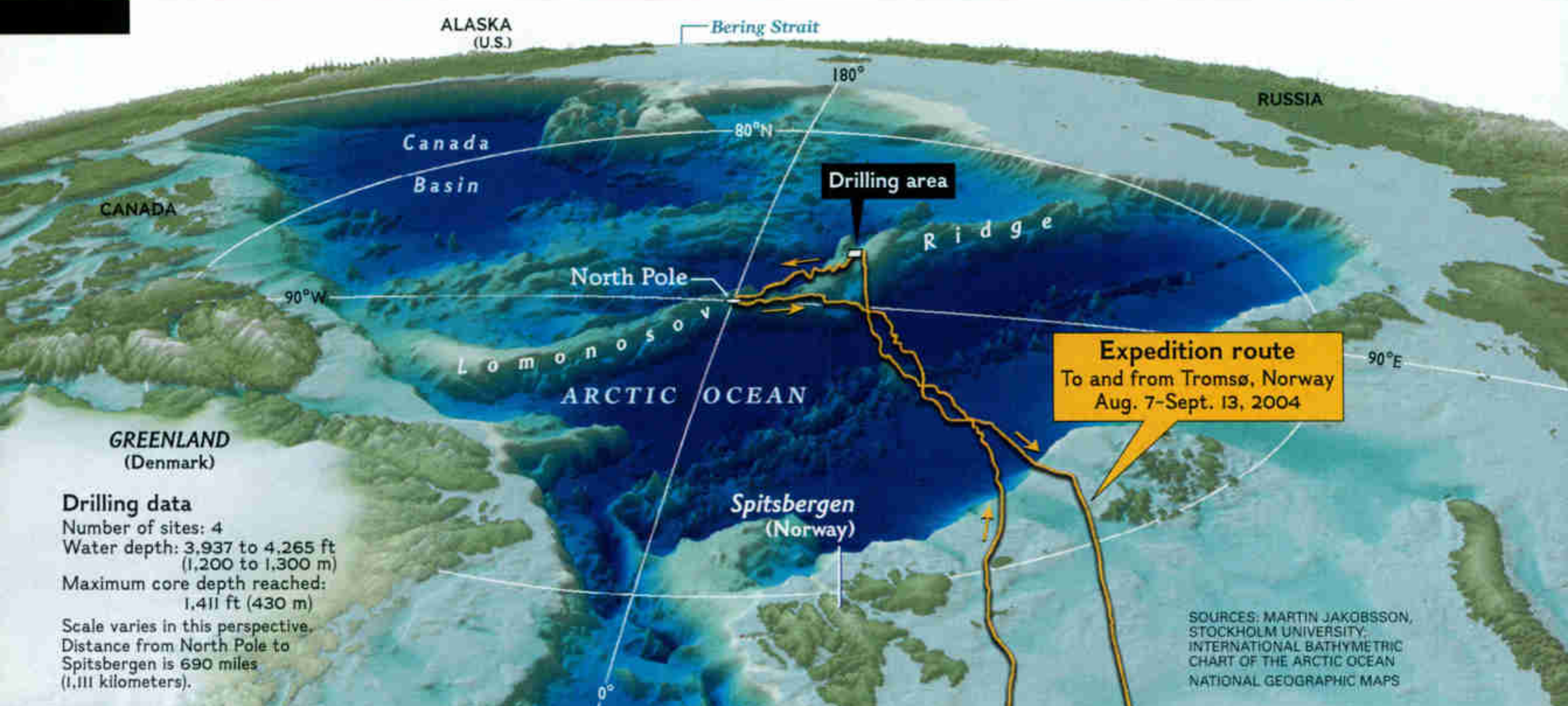
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More mysterious than the



Arctic's topography was its history. To read it would require drilling deep into seafloor sediment layers that record past conditions like the pages of a book. But controlling a drill plunging through thousands of feet of water on board a ship buffeted by pack ice was a daunting prospect.

"You have to protect the drill ship from the ice," says Kate Moran. An oceanographer at the University of Rhode Island, Moran served as co-leader—along with Jan Backman of Stockholm University—of the three-ship, European-funded Arctic Coring Expedition. One vessel, a Russian nuclear-powered behemoth (below, background) smashed the biggest floes, some a quarter mile wide or more and eight to ten feet thick, while another, a diesel icebreaker from Sweden, at center, fended off smaller bits.

The third icebreaker, foreground, carried the drill rig, which bored into the seafloor nearly 4,300 feet below (map) for nearly three weeks. "I don't think we can quite believe we did it!" Moran said after the ships docked in Norway last summer with 1,100 feet of samples spanning 55 million years.

The rotating drill bit had scrambled some of the samples, and about 600 feet below the seafloor, perhaps 30 million years of sediments—from 45 to 15 million years ago—were simply gone, perhaps scoured away eons ago by strong currents.

Below that gap, a window opened on a strange world. The very oldest sediments were riddled with the bat-winged fossils of a microscopic, warmth-loving marine alga called *Apectodinium*

(inset). Its presence signaled that the drillers had reached the time 55 million years ago when Earth experienced its warmest spell since the age of dinosaurs. Crocodiles basked in Greenland then, and Mediterranean warmth apparently reached the very top of the planet.

Slightly younger layers, 50 million years old, yielded an even bigger surprise. In the shipboard lab, a scientist examining samples through a microscope was baffled to see "stuff that looks like cow patties." But Henk Brinkhuis, a paleontologist from Utrecht University and an expert in tiny marine fossils, knew what it was: spores from *Azolla*—a fern that grows in fresh water.

Brinkhuis, who had done consulting work for the oil industry, remembered that crews drilling in seas bordering the Arctic had noticed a mysterious sprinkling of fern spores in 50-million-year-old rock. No one knew the source. Now Brinkhuis and his colleagues think they've found it: a once verdant Arctic Ocean.

Some expedition scientists believe the fern grew in river deltas or coastal lagoons, its spores

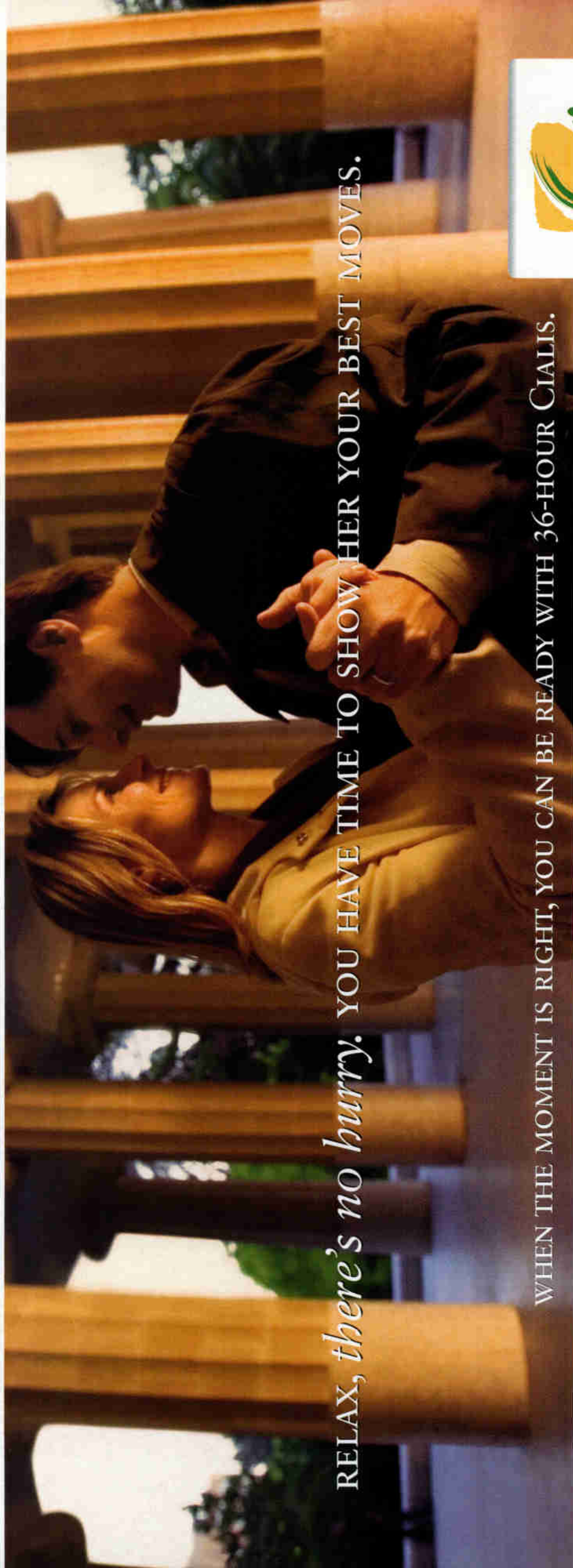


Cialis is not for everyone. If you take nitrates, often used for chest pain (also known as angina), or alpha-blockers (other than Flomax 0.4 mg once daily), prescribed for prostate problems or high blood pressure, do not take Cialis. Such combinations could cause a sudden, unsafe drop in blood pressure. Don't drink alcohol in excess (to a level of intoxication) with Cialis. This combination may increase your chances of getting dizzy or lowering your blood pressure. Cialis does not protect a man or his partner from sexually transmitted diseases, including HIV.

The most common side effects with Cialis were headache and upset stomach. Backache and muscle ache were also reported, sometimes with delayed onset. Most men weren't bothered by the side effects enough to stop taking Cialis. Although a rare occurrence, men who experience an erection for more than 4 hours (priapism) should seek immediate medical attention. Discuss your medical conditions and medications with your doctor to ensure Cialis is right for you and that you are healthy enough for sexual activity.

Cialis® is a registered trademark of Lilly ICOS LLC. Flomax® (tamsulosin HCl) is a registered trademark of its owner.

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Ask your doctor if prescription Cialis is right for you. See important safety information above and Patient Information on following page.

*Individual results may vary. Not studied for multiple attempts per dose. In clinical trials, Cialis was shown to improve, up to 36 hours after dosing, the ability of men with ED to have a single successful intercourse attempt.



Cialis®
(tadalafil) tablets

www.cialis.com

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Patient Information

CIALIS® (See-AL-iss) (tadalafil) tablets

Read the Patient Information about CIALIS before you start taking it and again each time you get a refill. There may be new information. You may also find it helpful to share this information with your partner. This leaflet does not take the place of talking with your doctor. You and your doctor should talk about CIALIS when you start taking it and at regular checkups. If you do not understand the information, or have questions, talk with your doctor or pharmacist.

What important information should you know about CIALIS?

CIALIS can cause your blood pressure to drop suddenly to an unsafe level if it is taken with certain other medicines. You could get dizzy, faint, or have a heart attack or stroke.

Do not take CIALIS if you:

- **take any medicines called “nitrates.”**
- **use recreational drugs called “poppers” like amyl nitrate and butyl nitrate.**
- **take medicines called alpha blockers, other than Flomax® (tamsulosin HCl) 0.4 mg daily.**

(See “Who should not take CIALIS?”)

Tell all your healthcare providers that you take CIALIS. If you need emergency medical care for a heart problem, it will be important for your healthcare provider to know when you last took CIALIS.

After taking a single tablet, some of the active ingredient of CIALIS remains in your body for more than 2 days. The active ingredient can remain longer if you have problems with your kidneys or liver, or you are taking certain other medications (see “Can other medications affect CIALIS?”).

What is CIALIS?

CIALIS is a prescription medicine taken by mouth for the treatment of erectile dysfunction (ED) in men.

ED is a condition where the penis does not harden and expand when a man is sexually excited, or when he cannot keep an erection. A man who has trouble getting or keeping an erection should see his doctor for help if the condition bothers him. CIALIS may help a man with ED get and keep an erection when he is sexually excited.

CIALIS does not:

- cure ED
- increase a man’s sexual desire
- protect a man or his partner from sexually transmitted diseases, including HIV. Speak to your doctor about ways to guard against sexually transmitted diseases.
- serve as a male form of birth control

CIALIS is only for men with ED. CIALIS is not for women or children. CIALIS must be used only under a doctor’s care.

How does CIALIS work?

When a man is sexually stimulated, his body’s normal physical response is to increase blood flow to his penis. This results in an erection. CIALIS helps increase blood flow to the penis and may help men with ED get and keep an erection satisfactory for sexual activity. Once a man has completed sexual activity, blood flow to his penis decreases, and his erection goes away.

Who can take CIALIS?

Talk to your doctor to decide if CIALIS is right for you. CIALIS has been shown to be effective in men over the age of 18 years who have erectile dysfunction, including men with diabetes or who have undergone prostatectomy.

Who should not take CIALIS?

- Do **not** take CIALIS if you:
- **take any medicines called “nitrates”** (See “What important information should you know about CIALIS?”). Nitrates are commonly used to treat angina. Angina is a symptom of heart disease and can cause pain in your chest, jaw, or down your arm. Medicines called nitrates include nitroglycerin that is found in tablets, sprays, ointments, pastes, or patches. Nitrates can also be found in other medicines such as isosorbide dinitrate or isosorbide mononitrate. Some recreational drugs called “poppers” also contain nitrates, such as amyl nitrate and butyl nitrate. Do not use CIALIS if you are using these drugs. Ask your doctor or pharmacist if you are not sure if any of your medicines are nitrates.
 - **take medicines called “alpha blockers”, other than Flomax® 0.4 mg daily.** Alpha blockers are sometimes prescribed for prostate problems or high blood pressure. If CIALIS is taken with alpha blockers other than Flomax® 0.4 mg daily, your blood pressure could suddenly drop to an unsafe level. You could get dizzy and faint.
 - **you have been told by your healthcare provider to not have sexual activity because of health problems.** Sexual activity can put an extra strain on your heart, especially if your heart is already weak from a heart attack or heart disease.
 - **are allergic to CIALIS or any of its ingredients.** The active ingredient in CIALIS is called tadalafil. See the end of this leaflet for a complete list of ingredients.

What should you discuss with your doctor before taking CIALIS?

Before taking CIALIS, tell your doctor about all your medical problems, including if you:

- **have heart problems** such as angina, heart failure, irregular heartbeats, or have had a heart attack. Ask your doctor if it is safe for you to have sexual activity.
- **have low blood pressure** or have high blood pressure that is not controlled
- **have had a stroke**
- **have liver problems**
- **have kidney problems or require dialysis**
- **have retinitis pigmentosa**, a rare genetic (runs in families) eye disease
- **have stomach ulcers**
- **have a bleeding problem**
- **have a deformed penis shape** or Peyronie’s disease
- **have had an erection that lasted more than 4 hours**
- **have blood cell problems** such as sickle cell anemia, multiple myeloma, or leukemia

Can other medications affect CIALIS?

Tell your doctor about all the medicines you take including prescription and non-prescription medicines, vitamins, and herbal supplements. CIALIS and other medicines may affect each other. Always check with your doctor before starting or stopping any medicines. Especially tell your doctor if you take any of the following:

- medicines called nitrates (See “What important information should you know about CIALIS?”)
- medicines called alpha blockers. These include Hytrin® (terazosin HCl), Flomax® (tamsulosin HCl), Cardura® (doxazosin mesylate), Minipress® (prazosin HCl) or Uroxatral® (alfuzosin HCl).
- ritonavir (Norvir®) or indinavir (Crixivan®)
- ketoconazole or itraconazole (such as Nizoral® or Sporanox®)
- erythromycin
- other medicines or treatments for ED

How should you take CIALIS?

Take CIALIS exactly as your doctor prescribes. CIALIS comes in different doses (5 mg, 10 mg, and 20 mg). For most men, the recommended starting dose is 10 mg. **CIALIS should be taken no more than once a day.** Some men can only take a low dose of CIALIS because of medical conditions or medicines they take. Your doctor will prescribe the dose that is right for you.

- If you have kidney problems, your doctor may start you on a lower dose of CIALIS.
- If you have kidney or liver problems or you are taking certain medications, your doctor may limit your highest dose of CIALIS to 10 mg and may also limit you to one tablet in 48 hours (2 days) or one tablet in 72 hours (3 days).

Take one CIALIS tablet before sexual activity. In some patients, the ability to have sexual activity was improved at 30 minutes after taking CIALIS when compared to a sugar pill. The ability to have sexual activity was improved up to 36 hours after taking CIALIS when compared to a sugar pill. You and your doctor should consider this in deciding when you should take CIALIS prior to sexual activity. Some form of sexual stimulation is needed for an erection to happen with CIALIS. CIALIS may be taken with or without meals.

Do not change your dose of CIALIS without talking to your doctor. Your doctor may lower your dose or raise your dose, depending on how your body reacts to CIALIS.

Do not drink alcohol to excess when taking CIALIS (for example, 5 glasses of wine or 5 shots of whiskey). When taken in excess, alcohol can increase your chances of getting a headache or getting dizzy, increasing your heart rate, or lowering your blood pressure.

If you take too much CIALIS, call your doctor or emergency room right away.

What are the possible side effects of CIALIS?

The most common side effects with CIALIS are headache, indigestion, back pain, muscle aches, flushing, and stuffy or runny nose. These side effects usually go away after a few hours. Patients who get back pain and muscle aches usually get it 12 to 24 hours after taking CIALIS. Back pain and muscle aches usually go away by themselves within 48 hours. Call your doctor if you get a side effect that bothers you or one that will not go away.

CIALIS may uncommonly cause:

- **an erection that won’t go away (priapism).** If you get an erection that lasts more than 4 hours, get medical help right away. Priapism must be treated as soon as possible or lasting damage can happen to your penis including the inability to have erections.
- vision changes, such as seeing a blue tinge to objects or having difficulty telling the difference between the colors blue and green.

These are not all the side effects of CIALIS. For more information, ask your doctor or pharmacist.

How should CIALIS be stored?

- Store CIALIS at room temperature between 59° and 86°F (15° and 30°C).
- **Keep CIALIS and all medicines out of the reach of children.**

General Information about CIALIS:

Medicines are sometimes prescribed for conditions other than those described in patient information leaflets. Do not use CIALIS for a condition for which it was not prescribed. Do not give CIALIS to other people, even if they have the same symptoms that you have. It may harm them.

This leaflet summarizes the most important information about CIALIS. If you would like more information, talk with your healthcare provider. You can ask your doctor or pharmacist for information about CIALIS that is written for health professionals.

For more information you can also visit www.cialis.com, or call 1-877-CIALIS1 (1-877-242-5471).

What are the ingredients of CIALIS?

Active Ingredient: tadalafil
Inactive Ingredients: croscarmellose sodium, hydroxypropyl cellulose, hypromellose, iron oxide, lactose monohydrate, magnesium stearate, microcrystalline cellulose, sodium lauryl sulfate, talc, titanium dioxide, and triacetin.

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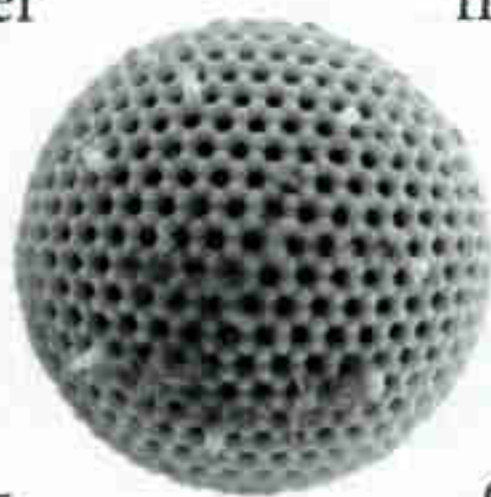
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drifting into the open ocean. But Brinkhuis argues that the Arctic Ocean itself must have been thick with vegetation. The spores were so plentiful that in the core samples “we have black layers that are virtually made of *Azolla*,” he says. He speculates that geologic forces closed the straits connecting other oceans to the Arctic, so runoff from rivers feeding into it pooled and formed a surface layer of fresh water where the fern could thrive.

Within a million years the fern vanished, maybe because the straits opened up again and flooded the Arctic with salt water, killing the fern. Soon after came the first hint of the frozen northern ocean of today. In samples as old as 47 million years, the scientists found dropstones, pebbles that froze to the bottom of ice floes in shallow water and fell off in mid-ocean. By 15 million years ago, layers of iceborne sand and gravel point to widespread freezing.

That’s earlier than scientists had thought. Many credited later events—rising mountains, shifting ocean currents—with lowering the global thermostat. Ice 15 million years ago, let alone 47 million years ago, calls for



HANNES VON DER FECHT (ABOVE); NALÂN KOÇ (INSET); MARTIN JAKOBSSON

Aboard ship, roughnecks lug a core sample from the Arctic floor. Henk Brinkhuis (bottom, at left) and Alexei Krylov study the samples for clues to the ancient ocean, such as a fossil diatom (inset).

another explanation, and a giant freshwater pool could be part of it, says Kate Moran.

Since fresh water is easier to freeze than salt, the ancient Arctic would have been “ripe for freezing,” she says. The ice, in turn, would have reflected sunlight, cooling the climate in a feedback effect that would have resulted in even more ice.

The fern may have been an accomplice. Scientists think a powerful greenhouse effect from atmospheric carbon dioxide ten times higher than today’s fueled the global warm spell 55 million years ago. Before ice could form, the carbon dioxide level had to drop. The fast-growing *Azolla* could have helped by sucking carbon from the air to build leaves and stems. As the ferns died, they would have sunk into deep waters, where scarce oxygen prevented their decay and kept the carbon locked up. “I’m really intrigued by the possibility that it was this little phase that turned the world into an icehouse,” Brinkhuis says.

It will take more study, and probably more drilling, to know exactly what caused the Arctic’s big chill. “After all, we’ve just

stuck one needle hole into this vast ocean,” says Nalân Koç of the Norwegian Polar Institute.

These days, as climate warms and the Arctic ice shrinks year by year, we may be undoing the change that began back in that warm, green Arctic 50 million years ago. Says Moran: “Understanding when and why the ice formed is even more important now that we humans are helping reverse the process.”

—Tim Appenzeller

Defrosting the Arctic

The far north may not have regained its old lushness, but signs of warming are everywhere.

Dwindling sea ice in the waters north of Alaska and Canada is threatening some sea life—and rekindling the dream of a Northwest Passage.

Plants thriving in the warmer climate are turning sparsely vegetated tundra into shrubland.

Fires and insect outbreaks fostered by warmer temperatures are assaulting northern forests.

TAKE TO THE ICE with Arctic researchers from another NGM story, “Northern Exposure,” at nationalgeographic.com/magazine/0401/feature6.





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ARCHAEOLOGY

The First Americans?

Discovery could rewrite history of human migration

Archaeologist Albert C. Goodyear thinks he's found some of the oldest artifacts in North America. If he's right, humans arrived in the New World tens of thousands of years earlier than previously thought.

In a chert quarry on the banks of the Savannah River (above), Goodyear and his team from the University of South Carolina have unearthed what appear to be human-chipped stone flakes and charred plants, possibly from a hearth. Carbon testing shows that the materials date back at least 50,000 years—to the Ice Age. This is far earlier than any previous evidence of humans in North America.

Goodyear's site, named Topper, is a layer cake of ancient remains, including scrapers and blades (above right) from a level estimated to be 16,000 to 20,000 years old.

Experts generally hold that the so-called Clovis people were the first to reach North America,

crossing from Siberia to Alaska on a land bridge about 14,000 years ago. Material goods uncovered at sites such as Meadowcroft in Pennsylvania, Monte Verde in Chile, and now Topper suggest humans arrived much earlier. But many scientists still view the finds with skepticism. Some say that what Goodyear calls artifacts are just ordinary rocks. Goodyear, too, once doubted pre-Clovis theories, but Topper converted him.

So what were humans doing in North America so long ago? "My guess is that they may have been coastal fisher-gatherers," says Goodyear. "The lower South was never glaciated. It may have been a pretty nice place for humans to come and hang out for a while."

In Goodyear's view, it's time for archaeologists to push past the "Clovis-first" model. Pre-Clovis is still controversial, "but the tide is turning," he says. "We need to dig deeper."

—Neil Shea

GEO NEWS

SPACE

■ **A meteorite has been found on Mars**—the first ever discovered on another planet. Mars rover Opportunity sent back images of the lone, shiny, basketball-size rock. Onboard sensors indicate it's likely composed of iron and nickel.

PRESERVATION

■ **Sir Ernest Shackleton's Antarctic expedition hut is being conserved** by the New Zealand-based Antarctic Heritage Trust. In 1908 Shackleton and his party wintered in the hut on the edge of the Ross Ice Shelf during their unsuccessful bid to be the first to reach the South Pole. Time and elements have damaged the hut and the hundreds of items they left behind in it.

HEALTH

■ **An unmade bed may help you breathe easier.** A study suggests that microscopic dust mites—implicated in some respiratory problems—are less likely to survive in unmade beds; exposure to air dehydrates the creatures. But warmth and moisture trapped in smoothed sheets may help dust mites thrive.

ANIMAL KINGDOM

■ **The platypus is stranger than we thought.** Researchers have found that the duck-billed, egg-laying Australian mammal has five pairs of chromosomes that determine its sex. Most mammals have just one pair. Even more intriguing, perhaps, is that platypus sex chromosomes show similarity to those of both mammals and birds, which are thought to have originated independently.



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JUERGEN SKARWAN, REUTERS/CORBIS

GEOGRAPHY

Surfing Brazil's *Pororoca*

River surfers ride the nearly endless wave

If surfing, the ancient art of wave riding, was transformed into music, it would be a blazing electric guitar riff, not a symphony. Surfers generally get just a fleeting stage on which to dance, as ocean waves that have traveled hundred of miles explode on shore in a matter of seconds. But not in Brazil.

There, like a mysterious beast, rises the wave known as the *pororoca*—the name means “mighty noise.” Twice a day around the new and full moons during the spring and fall, walls of chocolate-colored water rear up in several rivers in northern Brazil. The most powerful occurs in the Araguari, leaving a path of bankside destruction and flooding in its wake.

Scientists call such waves tidal bores. They are a regular phenomenon in many rivers around the world where a powerful incoming tide collides with a river's outflow.

For surfers, bores are anything

but boring. Ten-minute rides are common on a *pororoca*, which was first surfed in 1997. Brazilian surf star Alex “Picuruta” Salazar (above) has carved the wave for an astounding 37 minutes, riding more than seven miles.

The marathon wave comes with its own peculiar hazards. “There are caimans, piranhas, and poisonous snakes,” notes veteran California surfer Gary Linden, who has led four expeditions to Brazil in pursuit of the *pororoca*, “but it's one of the greatest experiences ever.”

—Joel Bourne

More Big Bores

Severn River, England Surfed since 1955, it surges some 20 miles through Gloucestershire.

Garonne and Dordogne Rivers, France Crowds gather to surf this bore, called the *mascaret*.

Qiantang River, China The world's largest tidal bore can muster a 20-foot-high wave.

WHAT IS IT?

A two-headed tortoise

Is it real? It is. The last of 14 hatchlings to emerge from tortoise breeder John Jones's latest brood of the endangered species, the two-headed Mediterranean spur-thighed tortoise hatched on August 24, 2004, in Dorchester, England.

What's its name? Solomon and Sheba, named after the biblical king and queen. But, Jones adds, “we sometimes call them ‘them,’ rather than ‘it.’”

What does it look like? When it first hatched, the tortoise was “a little bigger than a ten-pence piece,” says Jones—slightly more than an inch long. Its shell is slightly misshapen, and its back legs stick out to the side, so it “walks like it's doing the breaststroke.”

How does it act? Like most tortoises, this one “just eats and walks around,” says Jones. The right head is more dominant. “Sometimes, while one head is eating, the body will move away from the food because the other head sees food somewhere else.”

Are two heads rare? The aberration, which probably occurs at the embryonic stage, is uncommon but not unheard of. Recent reports include a two-headed tortoise hatched in South Africa in 2003.

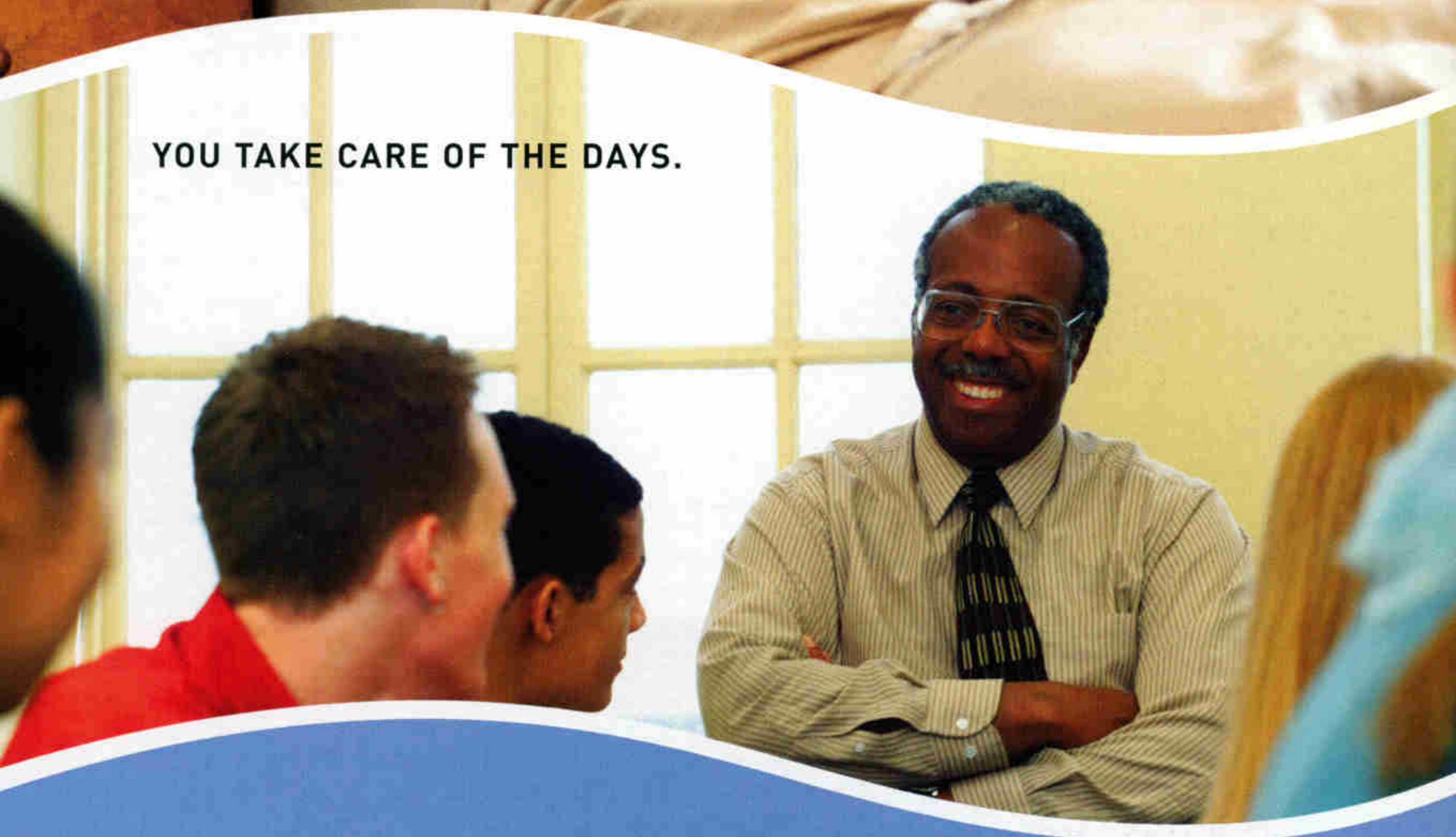
—Whitney Dangerfield



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AMBIEN is indicated for the short-term treatment of insomnia. There is a low occurrence of side effects associated with the short-term use of AMBIEN. The most commonly observed side effects in controlled clinical trials were drowsiness (2%), dizziness (1%), and diarrhea (1%). When you first start taking AMBIEN, use caution in the morning when engaging in activities requiring complete alertness until you know how you will react to this medication. In most instances, memory problems can be avoided if you take AMBIEN only when you are able to get a full night's sleep (7 to 8 hours) before you need to be active again. As with any sleep medication, do not use alcohol while you are taking AMBIEN. Prescription sleep aids are often taken for 7 to 10 days—or longer as advised by your doctor. All people taking sleep medicines have some risk of becoming dependent on the medicine.

Please see brief summary of prescribing information on adjoining page.

Ambien®

(zolpidem tartrate)

BRIEF SUMMARY

INDICATIONS AND USAGE

Ambien (zolpidem tartrate) is indicated for the short-term treatment of insomnia. Ambien has been shown to decrease sleep latency and increase the duration of sleep for up to 35 days in controlled clinical studies.

Hypnotics should generally be limited to 7 to 10 days of use, and reevaluation of the patient is recommended if they are to be taken for more than 2 to 3 weeks. Ambien should not be prescribed in quantities exceeding a 1-month supply (see *Warnings*).

CONTRAINDICATIONS

None known.

WARNINGS

Since sleep disturbances may be the presenting manifestation of a physical and/or psychiatric disorder, symptomatic treatment of insomnia should be initiated only after a careful evaluation of the patient. The failure of insomnia to remit after 7 to 10 days of treatment may indicate the presence of a primary psychiatric and/or medical illness which should be evaluated. Worsening of insomnia or the emergence of new thinking or behavior abnormalities may be the consequence of an unrecognized psychiatric or physical disorder. Such findings have emerged during the course of treatment with sedative/hypnotic drugs, including Ambien. Because some of the important adverse effects of Ambien appear to be dose related (see *Precautions and Dosage and Administration*), it is important to use the smallest possible effective dose, especially in the elderly.

A variety of abnormal thinking and behavior changes have been reported to occur in association with the use of sedative/hypnotics. Some of these changes may be characterized by decreased inhibition (eg, aggressiveness and extroversion that seemed out of character), similar to effects produced by alcohol and other CNS depressants. Other reported behavioral changes have included bizarre behavior, agitation, hallucinations, and depersonalization. Amnesia and other neuropsychiatric symptoms may occur unpredictably. In primarily depressed patients, worsening of depression, including suicidal thinking, has been reported in association with the use of sedative/hypnotics.

It can rarely be determined with certainty whether a particular instance of the abnormal behaviors listed above is drug induced, spontaneous in origin, or a result of an underlying psychiatric or physical disorder. Nonetheless, the emergence of any new behavioral sign or symptom of concern requires careful and immediate evaluation.

Following the rapid dose decrease or abrupt discontinuation of sedative/hypnotics, there have been reports of signs and symptoms similar to those associated with withdrawal from other CNS-depressant drugs (see *Drug Abuse and Dependence*).

Ambien, like other sedative/hypnotic drugs, has CNS-depressant effects. Due to the rapid onset of action, Ambien should only be ingested immediately prior to going to bed. Patients should be cautioned against engaging in hazardous occupations requiring complete mental alertness or motor coordination such as operating machinery or driving a motor vehicle after ingesting the drug, including potential impairment of the performance of such activities that may occur the day following ingestion of Ambien. Ambien showed additive effects when combined with alcohol and should not be taken with alcohol. Patients should also be cautioned about possible combined effects with other CNS-depressant drugs. Dosage adjustments may be necessary when Ambien is administered with such agents because of the potentially additive effects.

PRECAUTIONS

General

Use in the elderly and/or debilitated patients: Impaired motor and/or cognitive performance after repeated exposure or unusual sensitivity to sedative/hypnotic drugs is a concern in the treatment of elderly and/or debilitated patients. Therefore, the recommended Ambien dosage is 5 mg in such patients (see *Dosage and Administration*) to decrease the possibility of side effects. These patients should be closely monitored.

Use in patients with concomitant illness: Clinical experience with Ambien in patients with concomitant systemic illness is limited. Caution is advisable in using Ambien in patients with diseases or conditions that could affect metabolism or hemodynamic responses. Although studies did not reveal respiratory depressant effects at hypnotic doses of Ambien in normals or in patients with mild to moderate chronic obstructive pulmonary disease (COPD), a reduction in the Total Arousal Index together with a reduction in lowest oxygen saturation and increase in the times of oxygen desaturation below 80% and 90% was observed in patients with mild-to-moderate sleep apnea when treated with Ambien (10 mg) when compared to placebo. However, precautions should be observed if Ambien is prescribed to patients with compromised respiratory function, since sedative/hypnotics have the capacity to depress respiratory drive. Post-marketing reports of respiratory insufficiency, most of which involved patients with pre-existing respiratory impairment, have been received. Data in end-stage renal failure patients repeatedly treated with Ambien did not demonstrate drug accumulation or alterations in pharmacokinetic parameters. No dosage adjustment in renally impaired patients is required; however, these patients should be closely monitored (see *Pharmacokinetics*). A study in subjects with hepatic impairment did reveal prolonged elimination in this group; therefore, treatment should be initiated with 5 mg in patients with hepatic compromise, and they should be closely monitored.

Use in depression: As with other sedative/hypnotic drugs, Ambien should be administered with caution to patients exhibiting signs or symptoms of depression. Suicidal tendencies may be present in such patients and protective measures may be required. Intentional overdosage is more common in this group of patients; therefore, the least amount of drug that is feasible should be prescribed for the patient at any one time.

Information for patients: Patient information is printed in the complete prescribing information.

Laboratory tests: There are no specific laboratory tests recommended.

Drug interactions

CNS-active drugs: Ambien was evaluated in healthy volunteers in single-dose interaction studies for several CNS drugs. A study involving haloperidol and zolpidem revealed no effect of haloperidol on the pharmacokinetics or pharmacodynamics of zolpidem. Imipramine in combination with zolpidem produced no pharmacokinetic interaction other than a 20% decrease in peak levels of imipramine, but there was an additive effect of decreased alertness. Similarly, chlorpromazine in combination with zolpidem produced no pharmacokinetic interaction, but there was an additive effect of decreased alertness and psychomotor performance. The lack of a drug interaction following single-dose administration does not predict a lack following chronic administration.

An additive effect on psychomotor performance between alcohol and zolpidem was demonstrated.

A single-dose interaction study with zolpidem 10 mg and fluoxetine 20 mg at steady-state levels in male volunteers did not demonstrate any clinically significant pharmacokinetic or pharmacodynamic interactions. When multiple doses of zolpidem and fluoxetine at steady-state concentrations were evaluated in healthy females, the only significant change was a 17% increase in the zolpidem half-life. There was no evidence of an additive effect in psychomotor performance.

Following five consecutive nightly doses of zolpidem 10 mg in the presence of sertraline 50 mg (17 consecutive daily doses, at 7:00 am, in healthy female volunteers), zolpidem C_{max} was significantly higher (43%) and T_{max} was significantly decreased (53%). Pharmacokinetics of sertraline and N-desmethylsertraline were unaffected by zolpidem.

Since the systematic evaluations of Ambien in combination with other CNS-active drugs have been limited, careful consideration should be given to the pharmacology of any CNS-active drug to be used with zolpidem. Any drug with CNS-depressant effects could potentially enhance the CNS-depressant effects of zolpidem.

Drugs that affect drug metabolism via cytochrome P450: A randomized, double-blind, crossover interaction study in ten healthy volunteers between itraconazole (200 mg once daily for 4 days) and a single dose of zolpidem (10 mg) given 5 hours after the last dose of itraconazole resulted in a 34% increase in AUC_{0-24} of zolpidem. There were no significant pharmacodynamic effects of zolpidem on subjective drowsiness, postural sway, or psychomotor performance.

A randomized, placebo-controlled, crossover interaction study in eight healthy female volunteers between 5 consecutive daily doses of rifampin (600 mg) and a single dose of zolpidem (20 mg) given 17 hours after the last dose of rifampin showed significant reductions of the AUC (-73%), C_{max} (-58%), and $T_{1/2}$ (-36%) of zolpidem together with significant reductions in the pharmacodynamic effects of zolpidem.

Other drugs: A study involving cimetidine/zolpidem and ranitidine/zolpidem combinations revealed no effect of either drug on the pharmacokinetics or pharmacodynamics of zolpidem. Zolpidem had no effect on digoxin kinetics and did not affect prothrombin time when given with warfarin in normal subjects. Zolpidem's sedative/hypnotic effect was reversed by flumazenil; however, no significant alterations in zolpidem pharmacokinetics were found.

Drug/Laboratory test interactions: Zolpidem is not known to interfere with commonly employed clinical laboratory tests. In addition, clinical data indicate that zolpidem does not cross-react with benzodiazepines, opiates, barbiturates, cocaine, cannabinoids, or amphetamines in two standard urine drug screens.

Carcinogenesis, mutagenesis, impairment of fertility

Carcinogenesis: Zolpidem was administered to rats and mice for 2 years at dietary dosages of 4, 18, and 80 mg/kg/day. In mice, these doses are 26 to 520 times or 2 to 35 times the maximum 10-mg human dose on a mg/kg or mg/m² basis, respectively. In rats these doses are 43 to 876 times or 6 to 115 times the maximum 10-mg human dose on a mg/kg or mg/m² basis, respectively. No evidence of carcinogenic potential was observed in mice. Renal liposarcomas were seen in 4/100 rats (3 males, 1 female) receiving 80 mg/kg/day and a renal lipoma was observed in one male rat at the 18 mg/kg/day dose. Incidence rates of lipoma and liposarcoma for zolpidem were comparable to those seen in historical controls and the tumor findings are thought to be a spontaneous occurrence.

Mutagenesis: Zolpidem did not have mutagenic activity in several tests including the Ames test, genotoxicity in mouse lymphoma cells in vitro, chromosomal aberrations in cultured human lymphocytes, unscheduled DNA synthesis in rat hepatocytes in vitro, and the micronucleus test in mice.

Impairment of fertility: In a rat reproduction study, the high dose (100 mg base/kg) of zolpidem resulted in irregular estrus cycles and prolonged precoat intervals, but there was no effect on male or female fertility after daily oral doses of 4 to 100 mg base/kg or 5 to 130 times the recommended human dose in mg/m². No effects on any other fertility parameters were noted.

Pregnancy

Teratogenic effects: Category B. Studies to assess the effects of zolpidem on human reproduction and development have not been conducted.

Teratology studies were conducted in rats and rabbits.

In rats, adverse maternal and fetal effects occurred at 20 and 100 mg base/kg and included dose-related maternal lethargy and ataxia and a dose-related trend to incomplete ossification of fetal skull bones.

In rabbits, dose-related maternal sedation and decreased weight gain occurred at all doses tested. At the high dose, 16 mg base/kg, there was an increase in postimplantation fetal loss and underossification of sternebrae in viable fetuses.

This drug should be used during pregnancy only if clearly needed.

Nonteratogenic effects: Studies to assess the effects on children whose mothers took zolpidem during pregnancy have not been conducted. However, children born of mothers taking sedative/hypnotic drugs may be at some risk for withdrawal symptoms from the drug during the postnatal period. In addition, neonatal flaccidity has been reported in infants born of mothers who received sedative/hypnotic drugs during pregnancy.

Labor and delivery: Ambien has no established use in labor and delivery.

Nursing mothers: Studies in lactating mothers indicate that between 0.004 and 0.019% of the total administered dose is excreted into milk, but the effect of zolpidem on the infant is unknown.

The use of Ambien in nursing mothers is not recommended.

Pediatric use: Safety and effectiveness in pediatric patients below the age of 18 have not been established.

Geriatric use: A total of 154 patients in U.S. controlled clinical trials and 897 patients in non-U.S. clinical trials who received zolpidem were ≥60 years of age. For a pool of U.S. patients receiving zolpidem at doses of ≤10 mg or placebo, there were three adverse events occurring at an incidence of at least 3% for zolpidem and for which the zolpidem incidence was at least twice the placebo incidence (ie, they could be considered drug related).

Adverse Event	Zolpidem	Placebo
Dizziness	3%	0%
Drowsiness	5%	2%
Diarrhea	3%	1%

A total of 30/1,959 (1.5%) non-U.S. patients receiving zolpidem reported falls, including 28/30 (93%) who were ≥70 years of age. Of these 28 patients, 23 (82%) were receiving zolpidem doses >10 mg. A total of 24/1,959 (1.2%) non-U.S. patients receiving zolpidem reported confusion, including 18/24 (75%) who were ≥70 years of age. Of these 18 patients, 14 (78%) were receiving zolpidem doses >10 mg.

ADVERSE REACTIONS

Associated with discontinuation of treatment: Approximately 4% of 1,701 patients who received zolpidem at all doses (1.25 to 90 mg) in U.S. premarketing clinical trials discontinued treatment because of an adverse clinical event. Events most commonly associated with discontinuation from U.S. trials were daytime drowsiness (0.5%), dizziness (0.4%), headache (0.5%), nausea (0.6%), and vomiting (0.5%).

Approximately 4% of 1,959 patients who received zolpidem at all doses (1 to 50 mg) in similar foreign trials discontinued treatment because of an adverse event. Events most commonly associated with discontinuation from these trials were daytime drowsiness (1.1%), dizziness/vertigo (0.8%), amnesia (0.5%), nausea (0.5%), headache (0.4%), and falls (0.4%).

Data from a clinical study in which selective serotonin reuptake inhibitor- (SSRI) treated patients were given zolpidem revealed that four of the seven discontinuations during double-blind treatment with zolpidem (n=95) were associated with impaired concentration, continuing or aggravated depression, and manic reaction; one patient treated with placebo (n=97) was discontinued after an attempted suicide.

Incidence in controlled clinical trials

Most commonly observed adverse events in controlled trials: During short-term treatment (up to 10 nights) with Ambien at doses up to 10 mg, the most commonly observed adverse events associated with the use of zolpidem and seen at statistically significant differences from placebo-treated patients were drowsiness (reported by 2% of zolpidem patients), dizziness (1%), and diarrhea (1%). During longer-term treatment (28 to 35 nights) with zolpidem at doses up to 10 mg, the most commonly observed adverse events associated with the use of zolpidem and seen at statistically significant differences from placebo-treated patients were dizziness (5%) and drugged feelings (3%).

Treatment-emergent adverse experiences in placebo-controlled clinical trials: The following are treatment-emergent adverse events from U.S. placebo-controlled clinical trials. Data are limited to data from doses up to and including 10 mg. In short-term trials, events seen in zolpidem patients (n=685) at an incidence equal to 1% or greater compared to placebo (n=473) were: headache (7% vs 6% for placebo), drowsiness (2% vs 0%), dizziness (1% vs

0%), nausea (2% vs 3%), diarrhea (1% vs 0%), and myalgia (1% vs 2%). In long-term clinical trials, events seen in zolpidem patients (n=152) at an incidence of 1% or greater compared to placebo (n=161) were: dry mouth (3% vs 1% for placebo), allergy (4% vs 1%), back pain (3% vs 2%), influenza-like symptoms (2% vs 0%), chest pain (1% vs 0%), fatigue (1% vs 2%), palpitation (2% vs 0%), headache (19% vs 22%), drowsiness (8% vs 5%), dizziness (5% vs 1%), lethargy (3% vs 1%), drugged feeling (3% vs 0%), lightheadedness (2% vs 1%), depression (2% vs 1%), abnormal dreams (1% vs 0%), amnesia (1% vs 0%), anxiety (1% vs 1%), nervousness (1% vs 3%), sleep disorder (1% vs 0%), nausea (6% vs 6%), dyspepsia (5% vs 6%), diarrhea (3% vs 2%), abdominal pain (2% vs 2%), constipation (2% vs 1%), anorexia (1% vs 1%), vomiting (1% vs 1%), infection (1% vs 1%), myalgia (7% vs 7%), arthralgia (4% vs 4%), upper respiratory infection (5% vs 6%), sinusitis (4% vs 2%), pharyngitis (3% vs 1%), rhinitis (1% vs 3%), rash (2% vs 1%), and urinary tract infection (2% vs 2%).

Dose relationship for adverse events: There is evidence from dose comparison trials suggesting a dose relationship for many of the adverse events associated with zolpidem use, particularly for certain CNS and gastrointestinal adverse events.

Adverse events are further classified and enumerated in order of decreasing frequency using the following definitions: frequent adverse events are defined as those occurring in greater than 1/100 subjects; infrequent adverse events are those occurring in 1/100 to 1/1,000 patients; rare events are those occurring in less than 1/1,000 patients.

Frequent: abdominal pain, abnormal dreams, allergy, amnesia, anorexia, anxiety, arthralgia, asthenia, ataxia, back pain, chest pain, confusion, constipation, depression, diarrhea, diplopia, dizziness, drowsiness, drugged feeling, dry mouth, dyspepsia, euphoria, fatigue, headache, hiccup, infection, influenza-like symptoms, insomnia, lethargy, lightheadedness, myalgia, nausea, nervousness, palpitation, sleep disorder, vertigo, vision abnormal, vomiting.

Infrequent: abnormal hepatic function, agitation, arthritis, bronchitis, cerebrovascular disorder, coughing, cystitis, decreased cognition, detached, difficulty concentrating, dysarthria, dysphagia, dyspnea, edema, emotional lability, eye irritation, eye pain, falling, fever, flatulence, gastroenteritis, hallucination, hyperglycemia, hypertension, hypoesthesia, illusion, increased SGPT, increased sweating, leg cramps, malaise, menstrual disorder, migraine, pallor, paresthesia, postural hypotension, pruritus, scleritis, sleeping (after daytime dosing), speech disorder, stupor, syncope, tachycardia, taste perversion, thirst, tinnitus, trauma, tremor, urinary incontinence, vaginitis.

Rare: abdominal body sensation, abnormal accommodation, abnormal gait, abnormal thinking, abscess, acne, acute renal failure, aggressive reaction, allergic reaction, allergy aggravated, altered saliva, anaphylactic shock, anemia, angina pectoris, apathy, appetite increased, arrhythmia, arteritis, arthrosis, bilirubinemia, breast fibroadenosis, breast neoplasm, breast pain, bronchospasm, bullous eruption, circulatory failure, conjunctivitis, corneal ulceration, decreased libido, delusion, dementia, depersonalization, dermatitis, dysphasia, dysuria, enteritis, epistaxis, eructation, esophagospasm, extrasystoles, face edema, feeling strange, flushing, furunculosis, gastritis, glaucoma, gout, hemorrhoids, herpes simplex, herpes zoster, hot flashes, hypercholesterolemia, hyperhemoglobinemia, hyperlipidemia, hypertension aggravated, hypokinesia, hypotension, hypotonia, hypoxia, hysteria, impotence, increased alkaline phosphatase, increased BUN, increased ESR, increased saliva, increased SGOT, injection-site inflammation, intestinal obstruction, intoxicated feeling, lacrimation abnormal, laryngitis, leukopenia, lymphadenopathy, macrocytic anemia, manic reaction, micturition frequency, muscle weakness, myocardial infarction, neuralgia, neuritis, neuropathy, neurosis, nocturia, otitis externa, otitis media, pain, panic attacks, paresis, parosmia, periorbital edema, personality disorder, phlebitis, photopsia, photosensitivity reaction, pneumonia, polyuria, pulmonary edema, pulmonary embolism, purpura, pyelonephritis, rectal hemorrhage, renal pain, restless legs, rigors, sciatica, somnambulism, suicide attempts, tendinitis, tenesmus, tetany, thrombosis, tolerance increased, tooth caries, urinary retention, urticaria, varicose veins, ventricular tachycardia, weight decrease, yawning.

DRUG ABUSE AND DEPENDENCE

Controlled substance: Schedule IV.

Abuse and dependence: Studies of abuse potential in former drug abusers found that the effects of single doses of zolpidem tartrate 40 mg were similar, but not identical, to diazepam 20 mg, while zolpidem tartrate 10 mg was difficult to distinguish from placebo.

Sedative/hypnotics have produced withdrawal signs and symptoms following abrupt discontinuation. These reported symptoms range from mild dysphoria and insomnia to a withdrawal syndrome that may include abdominal and muscle cramps, vomiting, sweating, tremors, and convulsions. The U.S. clinical trial experience from zolpidem does not reveal any clear evidence for withdrawal syndrome. Nevertheless, the following adverse events included in DSM-III-R criteria for uncomplicated sedative/hypnotic withdrawal were reported at an incidence of $\leq 1\%$ during U.S. clinical trials following placebo substitution occurring within 48 hours following last zolpidem treatment: fatigue, nausea, flushing, lightheadedness, uncontrolled crying, emesis, stomach cramps, panic attack, nervousness, and abdominal discomfort. Rare post-marketing reports of abuse, dependence and withdrawal have been received.

Individuals with a history of addiction to, or abuse of, drugs or alcohol are at increased risk of habituation and dependence; they should be under careful surveillance when receiving any hypnotic.

OVERDOSAGE

Signs and symptoms: In European postmarketing reports of overdose with zolpidem alone, impairment of consciousness has ranged from somnolence to light coma, with one case each of cardiovascular and respiratory compromise. Individuals have fully recovered from zolpidem tartrate overdoses up to 400 mg (40 times the maximum recommended dose). Overdose cases involving multiple CNS-depressant agents, including zolpidem, have resulted in more severe symptomatology, including fatal outcomes.

Recommended treatment: General symptomatic and supportive measures should be used along with immediate gastric lavage where appropriate. Intravenous fluids should be administered as needed. Flumazenil may be useful. Respiration, pulse, blood pressure, and other appropriate signs should be monitored and general supportive measures employed. Sedating drugs should be withheld following zolpidem overdose. Zolpidem is not dialyzable.

The possibility of multiple drug ingestion should be considered.

Rx only

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My Seven



Medicines That Changed the World

John Swann *Historian, U.S. Food and Drug Administration*

John Swann earned a Ph.D. in pharmacy and the history of science from the University of Wisconsin, and has been at the FDA since 1989. His personal choices of history-making medicines appear “in more or less chronological order,” he says, “not in order of importance. I wouldn’t even try to rank medicines that way.” They’re all important—to someone.

1 Opium An important drug from a political, commercial, and cultural standpoint, opium was an early staple as a sedative and painkiller.

2 Smallpox vaccine Edward Jenner introduced a vaccine for smallpox made with the milder cowpox in 1798. In 1885 travelers from epidemic-stricken Montreal were vaccinated against the illness on board the train (right).

3 Salvarsan In 1910 Paul Ehrlich introduced the first drug crafted to attack a specific pathogen—the syphilis spirochete. Today he’s known as the father of chemotherapy for his revolutionary approach.

4 Insulin Before Frederick Banting and his colleagues isolated this hormone in the early 1920s, diets to



NGS PHOTOGRAPHER MARK THIESSEN (TOP LEFT); GRANGER COLLECTION, NEW YORK

treat diabetes essentially starved patients. Few other drugs have helped so many people so quickly.

5 Penicillin Discovered in 1928 but largely ignored, this antibiotic was resurrected during World War II to treat a range of what had been deadly infectious diseases.

6 Enovid Introduced as an oral contraceptive in the U.S.

in 1960, the “pill” had a revolutionary effect on the lives of millions. In 1970 it became the first medication to include a patient package insert conveying possible problems associated with its use. The notification was resisted by many health professionals at the time, but such disclosure is now standard practice.

7 Thalidomide During the late 1950s and early 1960s this sedative caused grave

birth defects in children whose mothers took it in pregnancy. This therapeutic disaster prompted radical changes in the way governments worldwide regulate medicines. Thalidomide reemerged in the 1990s as a treatment for complications of leprosy and other diseases.

WAR ON DISEASE

Former President Jimmy Carter talks about health issues and other challenges in the 21st century at nationalgeographic.com/ngm/0202.

Attention McDonald's Customers

McDonald's has searched for ways to reduce trans fatty acids (TFAs) in our food cooked with oil. On September 3, 2002, we announced a voluntary initiative to reduce TFAs by using a new cooking oil. When we were delayed in changing to the new cooking oil, we announced that delay on February 28, 2003.

Since then, we have changed the cooking process for Chicken McNuggets® and McChicken® and Crispy Chicken sandwiches, which reduced TFAs in those items. To avoid any confusion, McDonald's announces again that the cooking oil for its French fries, hash browns, and Filet-O-Fish® has not yet been changed. We continue to work on the initiative to reduce TFAs in our foods cooked with oil.

Please Read the Following Legal Notice.

The Superior Court of the State of California, County of Marin has preliminarily approved a settlement of two lawsuits against McDonald's. The lawsuits relate to TFA levels in McDonald's foods cooked with oil. The claims in those suits relate to: (i) the effectiveness of McDonald's communication about the status of its TFA initiative; (ii) McDonald's public statements about this initiative; (iii) the implementation or delay of this initiative; and (iv) TFA levels in McDonald's foods cooked with oil (collectively, the "Claims").

The lawsuits allege a violation of every state's consumer protection laws and fraud, breach of contract, negligence, breach of warranty, strict product liability, and battery. McDonald's has agreed to settle these lawsuits (the "Settlement"), but continues to deny vigorously that it violated any law.

What are the Principal Terms of the Settlement?

In exchange for a broad release of the Claims, McDonald's has agreed to, among other things:

- (a) donate \$7 million to the American Heart Association, a charity whose mission is to reduce disability and death from cardiovascular diseases and stroke, to be used exclusively for programs related to TFAs;
- (b) spend at least \$1.5 million notifying McDonald's customers about the delay in changing the cooking oil; and
- (c) pay legal fees, costs and expenses of Plaintiff's counsel in an amount not to exceed \$2 million, separate from and in addition to the amounts described above.

What are Your Legal Rights?

Remaining a Class Member

You are automatically a Class Member if you purchased or ate fried foods at any McDonald's restaurant in the U.S. after September 3, 2002. If you agree with the Settlement and wish to participate, you should do nothing.

If the Court grants final approval to the Settlement, you will be bound by the terms of the Settlement. You cannot sue McDonald's Corporation, any McDonald's franchisees, any McDonald's supplier, or any other McDonald's companies

(past, present, or future) for any of the Claims. You will have fully and finally released McDonald's of the Claims. The release to be given to McDonald's is broad and will release claims under federal and state law which you may have. If you believe you have claims, you should carefully consider whether you wish to remain a member of the Class, or whether you should exclude yourself from the Class.

Only persons who do validly exclude themselves from the Class may institute or continue to pursue Claims if the Court approves this Settlement.

Excluding Yourself

If you do not wish to participate, you may exclude yourself from the Class. Requests for exclusion and objections to the Settlement must be in writing and signed by you or your legal representative. If you choose to exclude yourself from the Class, your signed request must state your name and address and must be sent by mail to: Trans Fat Settlement, 3701 Sacramento Street, Box 500, San Francisco, CA 94118 postmarked no later than July 1, 2005.

Objecting to the Settlement

If you choose to object to the Settlement, you must file with the Clerk of the Court, with a copy to Trans Fat Settlement at the address below: (1) documents or an affidavit stating you are a member of the Class; and (2) a written statement of the basis for your objection(s). If you choose to appear at the Final Fairness Hearing described below, you must file a written notice of your intention to appear with the Clerk of the Court.

Approving the Settlement

A Final Fairness Hearing has been scheduled for August 24, 2005 at 9:30 a.m., in the Court to determine whether the proposed Settlement should be finally approved as fair, reasonable, and adequate. The Court will also hear and rule upon objections, if any, and determine whether and in what amount legal fees and expenses should be awarded.

The terms of the Settlement are set forth in detail in the parties' Stipulation of Settlement, which is available at the office of the Clerk of the Court, or on the Web site listed below.

For More Information About the Settlement,

Write: Trans Fat Settlement, 3701 Sacramento Street, Box 500, San Francisco, CA 94118

Visit: www.tfasettlement.com

By Order of the Honorable John A. Sutro Jr., Judge of the Superior Court of the State of California, Marin County.

ON ASSI

ON THE ROAD, IN THE FIELD,

INTERNATIONAL FOSSIL TRADE

Flying High, Siberian Style

Why getting there wasn't half the fun

The invitation sounded too good to resist: Follow Fyodor Shidlovskiy, a major Russian fossil dealer, to Siberia to meet his suppliers. But when they boarded a beat-up Russian cargo plane for the trip, writer **Lew Simons**, at center, in vest, interpreter **Ludmila Mekertycheva**, standing, and photographer **Lynn Johnson** realized there would be no heat or air-conditioning and no toilets—a challenge when the trip takes nearly ten hours. And instead of seats, the plane's 50 or so passengers were packed in on

backless benches. "Some of us stuck our noses into books and pretended we were in business class," says Lew.

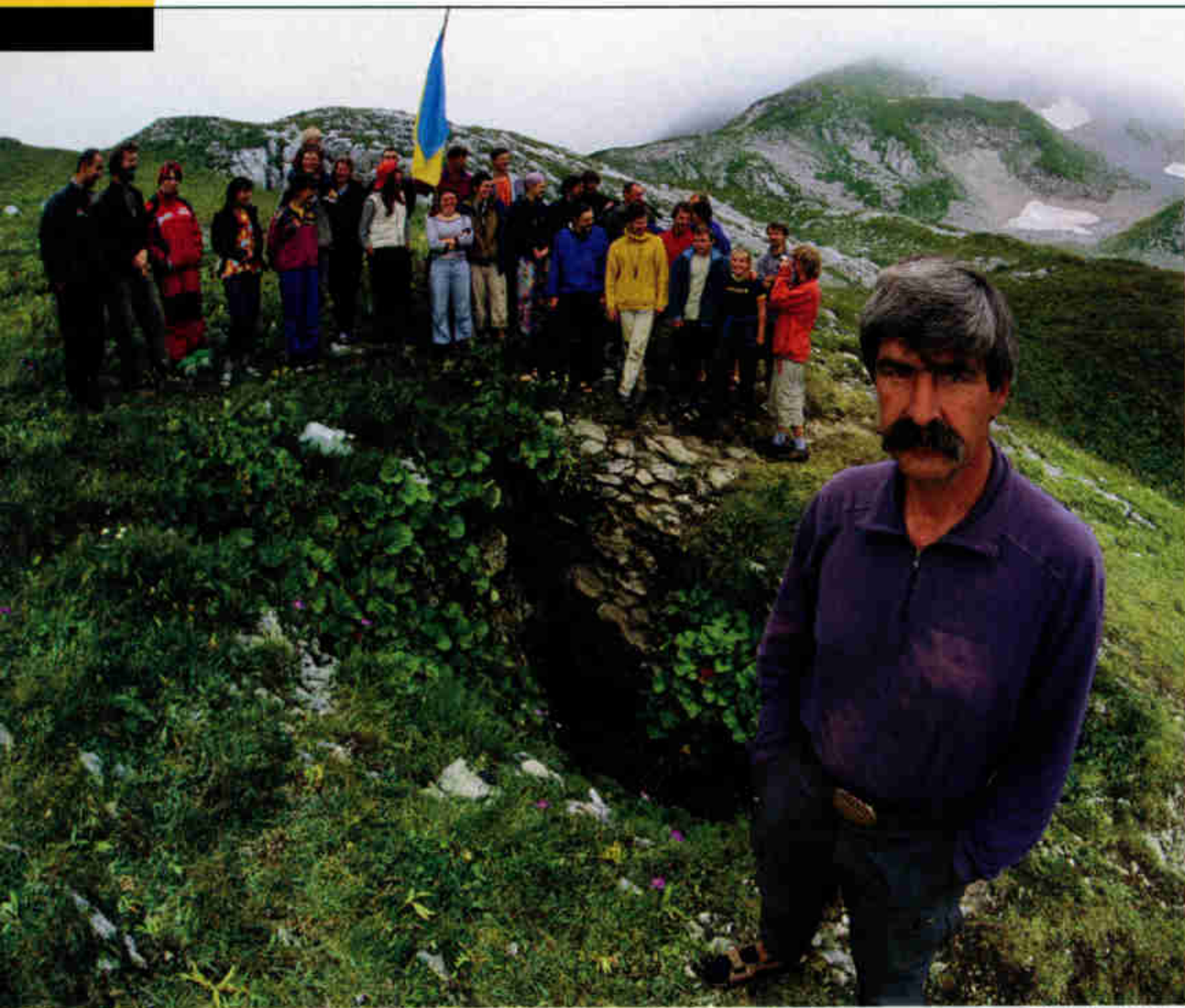
Their Siberian benchmates came better prepared, pulling out sacks of garlicky sausages, chocolate, and other treats to share, including a case of vodka. "In no time at all," says Lew, "we were a very merry band." When the plane finally shuddered to a stop north of the Arctic Circle, though, backs were aching, ears were ringing, and everyone was ready for the party to end.

GOVERNMENT

C O V E R I N G T H E W O R L D



LYNN JOHNSON



STEPHEN L. ALVAREZ

KRUBERA CAVE

An Extremely Long Way Down

If you happen to be hiking in the Caucasus Mountains, you could walk right past the opening to Krubera Cave and never know it was there. “Outside the cave it’s one of the most beautiful places,” says photographer **Stephen Alvarez**. “Inside is a living hell.”

An avid caver, Alvarez rappelled down the hole to cover author **Alexander Klimchouk’s** team as it explored Krubera, the world’s deepest cave. They descended 1,840 meters (6,037 feet), farther than any cavers had ever gone before.

Caving at those depths is taxing and dangerous. But, says Alexander (above, at right), “It’s my passion. It takes up all of my life.” He started caving at age 11. By the time he was 16, he’d published scientific papers on the geology of caves.

Alexander “is deeply obsessed

with finding the world’s deepest cave,” Stephen says. “He’s also a scientist, and everything he does is science driven.”

About five years ago, Alexander helped launch the Ukrainian Speleological Association’s Call of the Abyss project. The Krubera expedition is a part of that project. Participants (many of whom are pictured above) trained intensely: running ten kilometers (6.2 miles) a day, and working in construction or as window washers to keep strong and to accustom themselves to hanging from ropes for extended periods. The result was a cadre of cavers unfazed by 14 days within Krubera’s dark confines, where temperatures hovered at 36°F, and humidity reached 100 percent. Last October a second Call of the Abyss team descended 2,080 meters (1.29 miles) into Krubera.

This summer Alexander and other Call of the Abyss cavers head to Kuzgun Cave in eastern Turkey, where they hope to pass 2,000 meters. “Exploration to the ultimate limit,” says Alexander, “that is the goal.”

WORLDWIDE

CORAL REEF COLOR

Photographer **Tim Laman’s** aim was color. “I wanted to pursue the biological theme of the use of color by reef creatures,” he says. Location drives most underwater stories, but this time “it didn’t really matter to me where I shot the story.” Indonesia was a good choice: He’s been diving there for decades, and, as his photos reveal, the colors are spectacular.

POISON

As she tracked poison’s path, writer **Cathy Newman** learned the tricks of the trade from retired food taster Mathura Prasad (below, at left), who once served the lord of India’s Castle Mandawa. Though jobs for food tasters have dwindled, some heads of state still rate gastronomic guards. When George W. Bush visited Bangkok in 2003, a squadron of white lab mice under the jurisdiction of Thailand’s health ministry tested the U.S. President’s food. Cathy sought to interview the team, even submitting her questions in advance, but access was denied. Says Cathy, “The mice wouldn’t play.”



CARY WOLINSKY

TALES FROM THE FIELD Find more stories from our authors and photographers, including their best, worst, and quirkiest experiences, at nationalgeographic.com/magazine/0505.

Who Knew?

PHYSICS

Outside Player

Working in a patent office can be a good thing

Among the many reasons we revere Albert Einstein is the fact that he rewrote the laws of physics in his spare time. He had a day job as an examiner in the patent office in Bern, Switzerland.

He worked an eight-hour shift six days a week. He liked the paycheck. At night he'd wander around with a few friends and talk about physics.

Even after publishing four historic physics papers in 1905, his *annus mirabilis*, he stayed at the patent office. Einstein's miracle year should inspire everyone out there who, sitting in a coffee shop, sketching out some secret, elaborate, universe-altering theory on a napkin, dreams of someday being recognized as a genius rather than as a crank.

Could the young Einstein have performed his intellectual feats as an academic?

At the time, the field of theoretical physics was in its infancy, its practitioners often clashing

with the more established experimentalists. Einstein had been a spirited student with oodles of attitude, which comes in handy when you want to revise the basic laws of physics, but if he'd stayed in academia, some professors might have turned a deaf ear to a whippersnapper arguing that space and time aren't absolute.

Perhaps the patent office job helped in other ways. Einstein had to visualize and evaluate people's inventions based on drawings and specifications. His mind got a constant workout.

"Working on the final formulation of technological patents was a veritable blessing for me. It enforced many-sided thinking and also provided important stimuli to physical thought," Einstein wrote. Academia "places a young person under a kind of compulsion to produce impressive quantities of scientific publications—a temptation to superficiality."

Thomas Levenson, author of *Einstein in Berlin*, makes a key point: Although Einstein's miracle year occurred when he was an outsider, he eventually got the

first of many university jobs, and there on the inside, in 1909,

produced his greatest single work—the general theory of relativity, his definition of gravity.

The young Einstein was irrepressible whether he was an academic outsider at the patent office or an insider in Berlin. You might say he had, like a great basketball player, both an inside game and an outside game. Pop the jumper, or take it to the hoop. Maybe that analogy is too strange, too outside the box, but perhaps Einstein would approve.

—Joel Achenbach

WASHINGTON POST STAFF WRITER

Nobel Efforts

Einstein won a Nobel Prize, but not for the theory of relativity. Bitter nationalist sentiments of the post-World War I era played a role, but basically relativity proved to be too radical a concept for the Nobel committee. In 11 different years, Einstein was nominated only to be rejected. One Nobel committee member wrote: "Einstein must never receive a Nobel Prize even if the entire world demands it." The world did demand it, and Einstein got the 1921 Nobel—for his contributions to physics and for his 1905 paper on the photoelectric effect. He showed that light behaves not only as a wave but also as a stream of particles, or quanta. The committee directed Einstein not to mention relativity in his acceptance lecture. He did so anyway.

—Heidi Schultz

WEBSITE EXCLUSIVE For more on Einstein, and for links to Joel Achenbach's work, go to Resources at nationalgeographic.com/magazine/0505.

POISON

A 19th-century skull bottle would signal the lethality of its contents to someone fumbling for medicine in dim light.

TABLET (OPPOSITE) AND BOTTLE:
JOAN CABANISS COLLECTION



12

TOXIC TALES

Photographs by Cary Wolinsky

By Cathy Newman National Geographic Senior Writer

the *poison* paradox

too much can kill; a little can cure

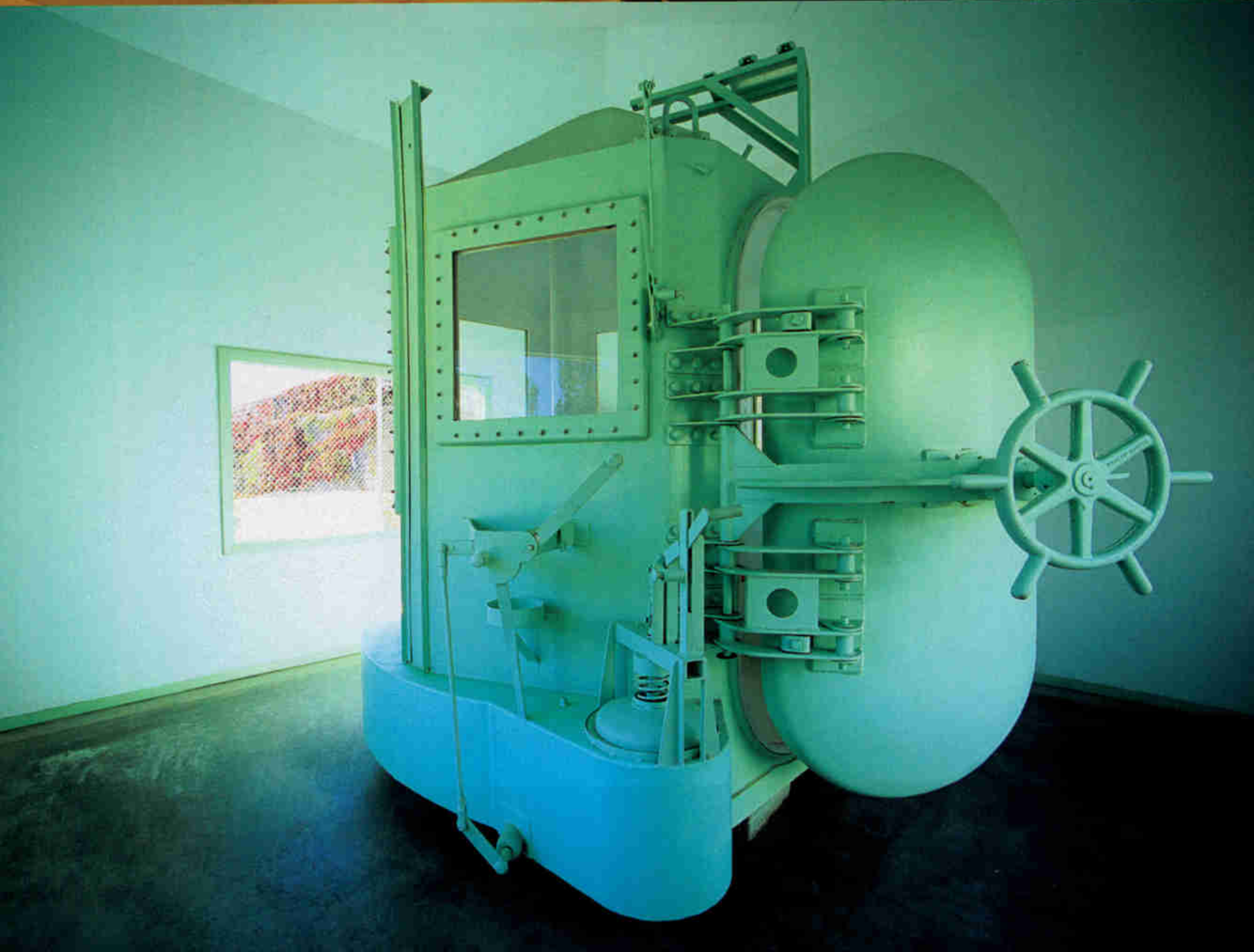
Bad things come in small packages. On August 14, 1996, Karen Wetterhahn, a toxicologist and professor of chemistry at Dartmouth College, spilled a drop, a tiny speck, of dimethylmercury on her left hand. Wetterhahn, tall, thin, intense, was an expert on how toxic metals cause cancer once they penetrate cell membranes. When she spilled the poisonous droplet in her lab, she thought nothing of it; she was wearing latex gloves. What she didn't know killed her.

The dimethylmercury was volatile enough to penetrate the glove. Five months later Wetterhahn began stumbling (*Continued on page 8*)

To build a better snake antivenin, take one chicken, inject small doses of snake venom, then harvest proteins from where they accumulate in the hen's eggs. Scientists at the Vittal Mallya Scientific Research Foundation in Bangalore, India, say the new method produces a less expensive antidote with fewer side effects than horse-serum-based antivenins currently in use.







{deadly doses}

HOLD A NICKEL IN YOUR HAND

Here's how many
lethal doses equal that
nickel's weight:

ANTHRAX
500,000,000

THALLIUM
5

1080 RAT
POISON
7

CYANIDE
25

ARSENIC
25

STRYCHNINE
50

NICOTINE
III

BOTULINUM
100,000,000

SOURCE: JOHN TRESTRIL,
DEVOS CHILDREN'S HOSPITAL
REGIONAL POISON CENTER,
GRAND RAPIDS, MICHIGAN

Fatal attractions: Jackie Bibby (right), holder of the world record for rattlesnake bathtub sitting, does it "for the rush." Eight prisoners were executed in the gas chamber now exhibited at the Museum of Colorado Prisons in Cañon City (left). Police at the U.S. Capitol in Washington, D.C., conduct a drill simulating release of a hazardous substance (left, above).



into doors and slurring words. After three weeks in a hospital, she slipped into a coma.

"I went to see her, but it wasn't the kind of coma I'd expected," recalled Diane Stearns, one of her postdoctoral students, now a professor of chemistry herself. "She was thrashing about. Her husband saw tears rolling down her face. I asked if she was in pain. The doctors said it didn't appear that her brain could even register pain."

Karen Wetterhahn died five months later. She was 48 years old, a wife and mother of two. The mercury had devoured her brain cells "like termites eating away for months," one of her doctors said. How could such a brilliant, meticulous, world-class toxicologist come to such an end?

"Only lion tamers are killed by lions," said Kent Sugdan, one of her postdoctoral fellows.

Poison is a stealth killer, effective in minuscule amounts, often undetectable. It's the treachery in the arsenic-tainted glass of wine. The fatal attraction: Snow White's poison apple, the death-defying art of the snake handler, the Japanese roulette practiced by those who eat fugu. Without poison, comic book superheroes and villains in plays and movies would be considerably duller. Spiderman exists by the grace of a radioactive spider bite. The rise of the Teenage Mutant Ninja Turtles can be traced to their fall (as pet turtles) into a sewer along with a container of toxic materials. Laertes used a poison-dipped sword to kill Hamlet, and Claude Rains's nasty mother kept sneaking poison drops into Ingrid Bergman's drinks in the Hitchcock thriller *Notorious*.

You might say that a toxicologist studies substances that lead to death. But toxicology is also about life. What can kill, can cure. Said Paracelsus, a 16th-century German-Swiss physician and alchemist: "All substances are poisons; there is

none which is not a poison. The right dose differentiates a poison and a remedy." Poison is in the dose. Toxicology and pharmacology are intertwined, inseparable, a Jekyll-Hyde duality. A serpent coiled around a staff symbolizes Asclepius, the Greek god of medicine.

Consider arsenic, the poison of kings and king of poisons. Arsenic exploits certain pathways in our cells, binds to proteins, and creates molecular havoc. Small amounts taken over a

long stretch produce weakness, confusion, paralysis. Take less than a tenth of an ounce at once, and the classic signs of acute arsenic poisoning ensue: nausea, vomiting, diarrhea, low blood pressure, then death.

Because it is colorless, tasteless, and odorless, arsenic was the poison of choice for the Borgias, the Italian Renaissance family skilled at artful murder, as well as for Hieronyma Spara, a 17th-century Roman entrepreneur who ran a school that taught wealthy young wives how to dispatch their husbands and become wealthy young widows. Arsenic, the *poudre de succession*, powder of succession, helped ambitious princes secure thrones. Fed in small amounts to a wet nurse, the poison could be expressed in breast milk and kill infant rivals.

From death to life: In the fifth century B.C., Hippocrates used arsenic to treat ulcers. It became an ingredient in Fowler's solution, created in 1786 and used for more than 150 years to treat every-

thing from asthma to cancer. In 1910 an arsenic compound became the first effective remedy for syphilis (later to be replaced by penicillin). Arsenic derivatives are still used to treat African sleeping sickness. In 1890 William Osler, founder of modern medical education, pronounced arsenic the best drug for leukemia, and today it remains an effective chemotherapy agent for acute forms of the disease.



arsenic poison or drug?

"It's both," says
Joshua Hamilton,
professor of toxicology
and pharmacology
at Dartmouth.

"It depends: Are you talking
to a Borgia, or are you talking
to a physician?"

So is arsenic a poison or a drug?

"It's both," says Joshua Hamilton, professor of toxicology and pharmacology at Dartmouth. "It depends: Are you talking to a Borgia, or are you talking to a physician?"

Poisons surround us. It's not just too much of a bad thing like arsenic that can cause trouble, it's too much of nearly anything. Too much vitamin A, hypervitaminosis A, can cause liver damage. Too much vitamin D can damage the kidneys. Too much water can result in hyponatremia, a dilution of the blood's salt content, which disrupts brain, heart, and muscle function.

Even oxygen has a sinister side. "Oxygen is the ultimate toxin," says Michael Trush, a toxicologist at Johns Hopkins Bloomberg School of Public Health. Oxygen combines with food to produce energy, but our bodies also produce oxygen radicals—atoms with an extra electron that damage biomolecules, DNA, proteins, and lipids. "We are oxidizing all the time," says Trush. "The biochemical price of breathing is aging." Which is to say, we rust.

As if everyday poisons aren't enough to angst over, there are nature's more exotic hazards. It's a jungle out there. There are 1,200 kinds of poisonous marine organisms, 700 poisonous fish, 400 venomous snakes, 60 ticks, 75 scorpions, 200 spiders, 750 poisons in more than 1,000 plant species, and several birds whose feathers are toxic when touched or ingested.

Given the treachery of the world, why don't more of us die of poisoning? Because our bodies are designed to protect us from both natural and man-made toxins. The first line of defense, skin, is made of keratin—so waterproof, tough, and tightly woven that only the smallest and most fat-soluble molecules can get through. Our senses warn us of noxious substances; if they fail there is vomiting as backup. Finally, there is the liver, which turns fat-soluble poisons into water-soluble wastes that can be flushed out through our kidneys. The balance tilts over to toxicity only when we step over the threshold of dosage.

Mike Gallo, a toxicologist, knows the principle of threshold from the inside out. Literally. Gallo, a hyper-caFFEinated personality wrapped in a wiry frame, is an associate director at the Cancer Institute of New Jersey in New Brunswick. In February 2004, at 64, he was diagnosed with non-Hodgkin's lymphoma. Two

weeks later he became both toxicologist and patient at the cancer institute. His oncologist put him on a four-month intravenous diet of toxins, also known as chemotherapy, and he began treatment in a clinic four floors down from his office.

The ingredients of his cocktail included cytoxan, adriamycin, vincristine, prednisone, and Retuxan—toxic enough to cause side effects ranging from vomiting, diarrhea, and weight loss, to liver, heart, and bladder damage, to death from overwhelming infection due to a depressed immune system. In addition, as Gallo will cheerfully tell you, "Almost all cancer drugs are carcinogenic in their own right."

On the other hand, he says, "The moment they stuck the needle in my vein, I felt relief. I thought, They got the son of a bitch."

Gallo was lucky. His luxuriant mop of red hair fell out, and he took on the alien look of chemotherapy. But fatigue and the typical drop in blood-cell count aside, he continued working through the treatment.

"I did just fine," he says, "but in the room right next to me is the same person, the same age, the same physique, and he's getting the stuffing kicked out of him. Why? My drug-metabolizing enzymes must be slightly different from his."

It's these pieces of toxicology—the matter of difference, the question of how much or how little, the wavering line between killing and curing—that Gallo loves so much as a scientist. They are the heart of toxicology and thus of poison. "Toxicology gives you the chance to understand biology," he says.

Toxicology also saved his life. Six months and thousands of milligrams of toxic drugs later, Gallo's doctor gave him the all-clear. The lymphoma is in remission.

The tale of two toxicologists ends tragically for one, happily for the other. Karen Wetterhahn lost her life to poison. Michael Gallo owes his life to it. "I dodged a lethal bullet, thanks to a series of well-placed bullets," Gallo says. "I could have been a dead man. Thank God for toxicity." ●

TOXIC TALES WIRED See Cary Wolinsky's behind-the-scenes video of a fugu chef carving up puffer fish to separate the deadly from the delicious; listen to the virtuosity of a pianist saved from incapacitating illness by one of the world's most lethal poisons at nationalgeographic.com/magazine/0505.

the curious case of *napoleon b.*

{1} *It's a game of Clue and historical whodunit all in one.*

The victim, Napoleon Bonaparte, died on May 5, 1821, on St. Helena, in exile after his defeat at Waterloo. An autopsy performed the next morning revealed perforation of the stomach due to an ulcer, possibly cancerous. The real cause of death? In dispute ever since. Some theories:

political murder

Murdered by arsenic poisoning, according to Ben Weider, founder of the

International Napoleonic Society and head of a huge Canada-based body-building empire. Weider has relentlessly sought the cause of Napoleon's death for more than four decades and has poured considerable resources into the quest. In his view, Napoleon was poisoned by the British and by French royalists, who wanted him out of the way once and for all. Weider offers as the

centerpiece of his hypothesis the hair analysis done by Pascal Kintz, a French toxicologist at the Legal Medicine Institute of Strasbourg. Kintz subjected samples of Napoleon's hair to a sophisticated technique known as nanosecondary ion mass spectrometry, which confirmed the long-term presence of arsenic. Kintz steps back from saying how or why the arsenic was there, but Weider is convinced that "the poisoning of Napoleon was planned and deliberate. Anything else is hogwash."

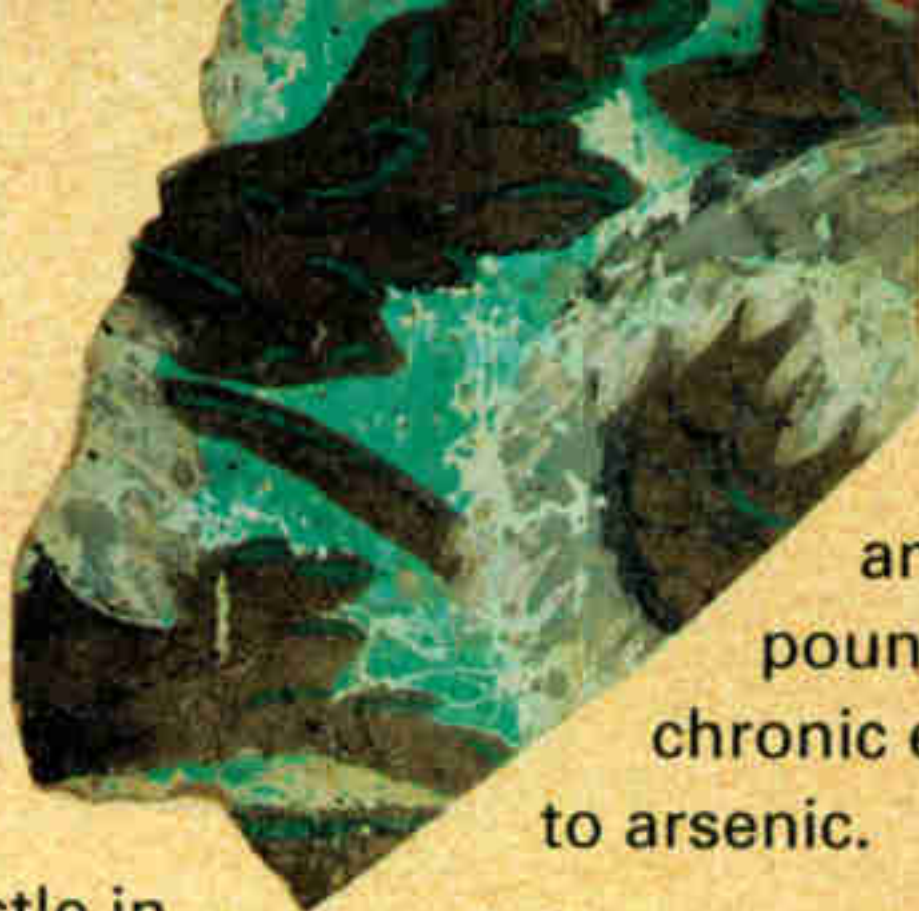


environmental poisoning

Poisoned by his wallpaper, theorizes David Jones, an immunologist at the University of Newcastle in England. The wallpaper at Longwood House, where Napoleon lived his last years, was painted with Scheele's green, an arsenic compound called copper arsenide. When attacked by certain molds, possibly present in the damp environment of St. Helena, arsenic would have been released into the air. In the late 1950s Clare Boothe Luce, the American ambassador to Italy, was diagnosed with arsenic poisoning caused by paint chips falling from the stucco roses on her bedroom ceiling.

malpractice

Killed by his doctors, says Steven Karch, a cardiac pathologist in Berkeley, California. Napoleon's doctors gave him large doses of purgatives including tarter emetic and, the day before his death, a massive dose of mercurous chloride, called calomel. The medications threw Napoleon's electrolytes into total disarray, Karch says, disrupting his heartbeat and resulting in cardiac arrest. In pathologist terms, the immediate cause of Napoleon's death was cardiac arrhythmia precipitated by



medical negligence and compounded by chronic exposure to arsenic.

disease

Cancer and ulcers as reported in the autopsy, says Jean Tulard, the preeminent Napoleon historian in France.

Tulard remains unconvinced by Kintz's hair analysis. In his estimation the provenance of the hair—whether it really belonged to Napoleon or not—is one of many problems standing in the way of definitive proof. "There are more samples of Napoleon's hair than relics of the Cross," he scoffs.

Above all, Tulard discounts the poisoning theory on the grounds that no one has yet found anything linking Hudson Lowe, the British governor-general of St. Helena—or anyone else for that matter—to any plot against Napoleon's life. "A bogus discussion," he says, "even if it is important to know how he died."

revenge

"One of my ancestors did it," says François de Candé-Montholon with a whiff of pride. ("I'm an aristocrat. Aristocrats don't like revolution, and Napoleon made revolution.")

Candé-Montholon's

great-great-great-great-grandfather, the Count of Montholon, was stationed with Napoleon on St. Helena. Napoleon had an affair—and fathered a child—with the count's wife. The count, it is observed, had charge of Napoleon's wine cellar and food. Could he, motivated by revenge, have poisoned the wine?

no conclusion . . .

"Everyone is right, and no one is right," says Paul Fornes, a forensic pathologist at the Hospital Georges Pompidou in Paris. Fornes has reviewed the 1821 autopsy report and other historical records and concludes:

"Napoleon may have died with cancer, but he didn't die of cancer."

Likewise he says that although the hair analysis indicates the presence of arsenic, no one can say if he was intentionally given the arsenic (or if it killed him). In Fornes's opinion the accusation of murder by poisoning would never fly in a court of law.

Believe what you will.

"We have left the world of history and science behind," says Jean-François Lemaire, a doctor and French historian, disdaining the circus (press conferences! newspaper stories!) surrounding the debate. "We are now in the world of entertainment." Or perhaps, as the French would say, it's a case of *couper les cheveux en quatre*—splitting hairs. ●



one bad move and you're

snakebit!

{2}

DURING A ONE-MAN WILDLIFE SURVEY ON A DESERTED FLORIDA BARRIER ISLAND, HERPETOLOGIST BRUCE MEANS FINDS HIS FAVORITE VENOMOUS REPTILE.*

HE KNOWS BETTER, BUT TRIES TO CAPTURE THE RATTLER WITH A STICK....

DEFENDING ITSELF, THE SNAKE STRIKES!


IT'S ONLY A PINPRICK ON HIS FINGER, BUT BRUCE KNOWS THE VENOM WILL START WORKING WITHIN SECONDS.

TISSUES BREAK DOWN AS ENZYMES IN THE VENOM ATTACK.


* EASTERN
DIAMONDBACK

RATTLESNAKE

(CROTALUS ADAMANTEUS)



AS TOXINS WREAK
HAVOC, BRUCE
CRAWLS TO FIND
HELP BEFORE
IT'S TOO LATE.



BLOOD AND OTHER
FLUIDS BEGIN TO
LEAK INTO HIS
TISSUES. HIS
BLOOD IS LOSING
ITS ABILITY
TO CLOT.
WILL HE DIE?

HE MADE IT.

BRUCE MANAGED TO REACH A HOSPITAL—AND SURVIVED. BUT THE EASTERN DIAMOND-BACK STILL CLAIMS AN OCCASIONAL LIFE. VENOM STRENGTH VARIES DEPENDING ON THE SNAKE'S AGE, WHEN IT LAST ATE, THE TIME OF DAY THE STRIKE OCCURS, HOW DEEPLY THE FANGS PENETRATE, AND HOW MUCH VENOM IS INJECTED.



WORST-CASE SCENARIO:

CIRCULATORY FAILURE, SHOCK, MASSIVE TISSUE NECROSIS, AND INTERNAL AND EXTERNAL BLEEDING LEAD TO DEATH. MEDICAL HELP IS THE KEY, BUT SOME WAIT TOO LONG BEFORE SEEKING TREATMENT. OTHERS, OFTEN CHILDREN, ARE JUST NOT ROBUST ENOUGH TO WITHSTAND THE POISON'S LETHAL EFFECT.

{3} “I was lost,” Leon Fleisher says, and 40 years later you can still feel the suffocating despair. One of the world’s premier concert pianists, Fleisher was talking about the aftermath of a day in 1965 when the career so carefully nurtured (his first public recital at 8; a performance with the New York Philharmonic in Carnegie Hall at 16) unexpectedly ended.

Fleisher, a man with a spirit as expansive as a Beethoven symphony, sits in the music room of his Baltimore home. Twin Steinway grands nest together; on one there are photographs of a young, gangly Leonard Bernstein and of George Szell, the legendary maestro of the Cleveland Orchestra (“looking cold as ever,” Fleisher notes). The conversation drifts to the day in Cleveland’s Severance Hall when Szell rehearsed Fleisher and the orchestra in final preparation for a tour of the Soviet Union. “It was the height of the Cold War. We were going to show the Russians

concerto in b for botox *e* piano

what music was all about,” Fleisher recalls. “I had noticed the fourth and fifth fingers on my right hand curling under involuntarily. I figured, Wow, I better work harder. I did. It got worse. George noticed too.”

When rehearsal ended, Szell called Fleisher to his study. “I don’t think you should come on tour,” he said. That was it. Fleisher was 37. His life had evaporated.

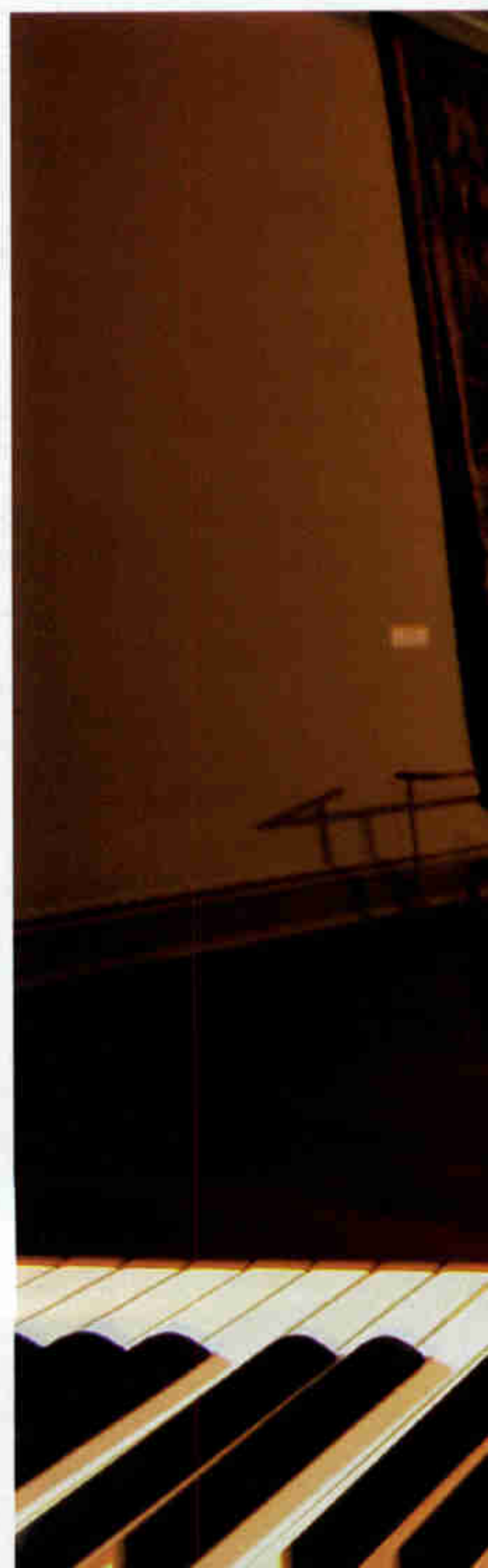
There were doctors: orthopedists, neurologists, a hand surgeon, psychiatrists. There were injections, x-rays, medications, acupuncture, aromatherapy. All failed. All useless. “It was as if my hand had been taken over by aliens,” he says. “It was not under my control.”

A career ruined. A marriage wrecked. Thoughts, even, of suicide.

“Finally I realized my connection to music was stronger than just as a two-handed piano player. I started conducting, playing the left-handed repertory, and teaching at the Peabody Conservatory.” Yet the pain of the missing piece of his life persisted. “I taught and conducted and every bloody day I tested this hand.” He lifts the offending hand and demonstrates how the fingers curled under like claws.

There was, it should be noted, a brief respite in 1981 when the condition seemed to improve. Fleisher played at the opening of the Meyerhoff Hall in Baltimore. “I managed to get through,” he recalls, “but just barely. Afterward I broke down backstage. A grown man weeping. . . .”

After decades a diagnosis emerged. Fleisher was afflicted with focal dystonia, a misfiring of the brain that causes muscles to contract into abnormal, and sometimes painful, positions. The disorder often strikes those who depend on small motor skills: musicians, writers, surgeons. At last relief seemed possible. He was referred to a clinical trial at the National Institutes of Health, where botulinum toxin was being tested as a remedy for the disabling contractions.



Botulinum toxin is produced from the bacterium *Clostridium botulinum*, one of the most poisonous substances known. A gram of botulinum toxin, if dispersed and ingested, could kill 20 million people. The toxin produces a protein that blocks the release of acetylcholine, a transmitter that tells a muscle to contract. In extremely dilute form the poison, delivered in the drug Botox, has proved effective and safe in medical applications ranging from the softening of wrinkles, to the relief of migraines, to a cure for crossed eyes, to a treatment for the spastic contractions of multiple sclerosis and cerebral palsy.

Botulinum toxin relieves symptoms without curing the condition, so Fleisher receives an injection every six months or so. But the six-month miracle is a miracle no less.

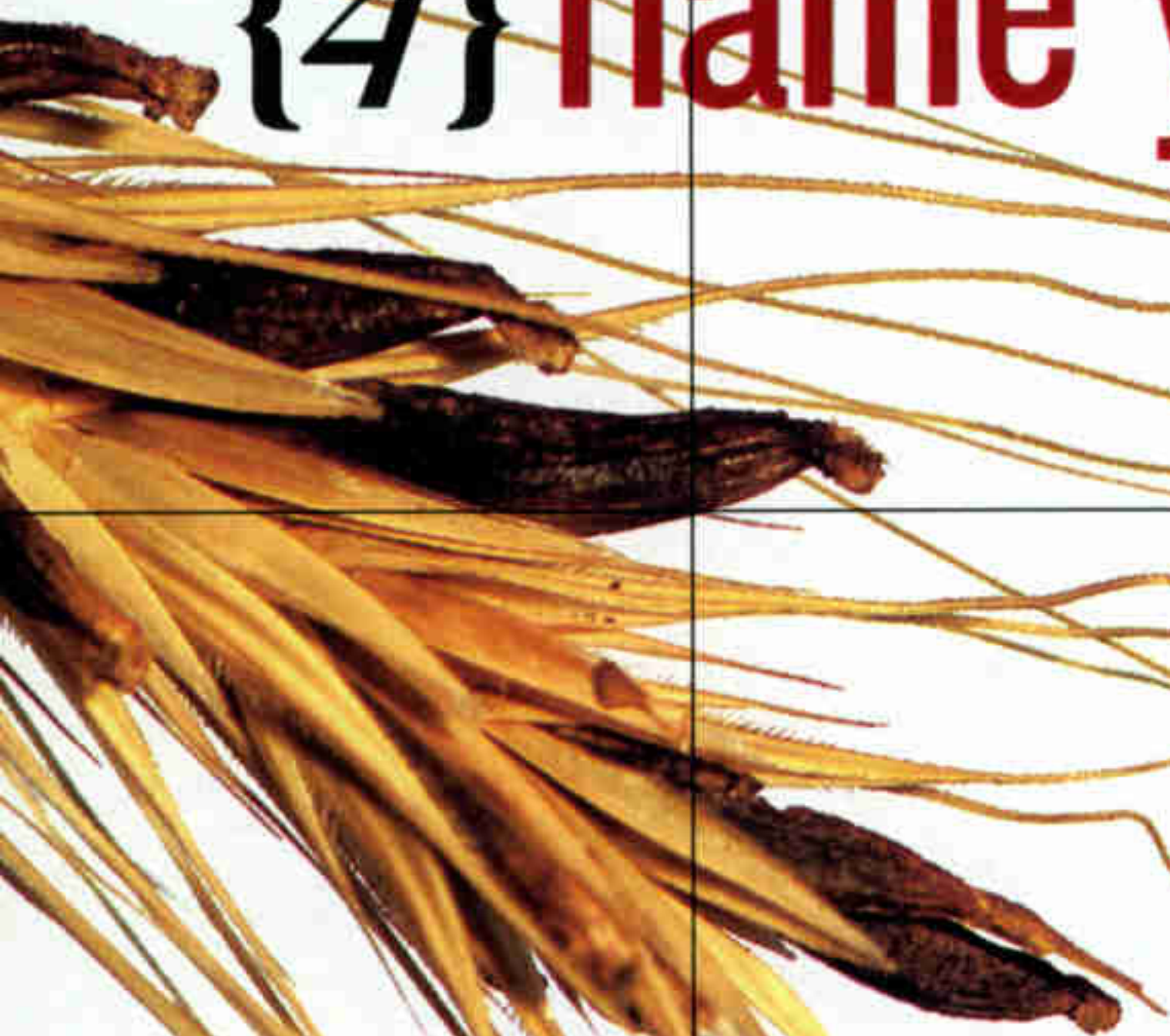
"I have had eight, maybe nine lives," Fleisher says. For each there is cause to celebrate, but maybe most of all for the ninth. He is performing and touring again, and recently released his first two-handed recording in 40 years.

Artur Schnabel, Fleisher's mentor, whose teacher's teacher was Beethoven himself, once said that life is about ascendancy. The only thing that grows down is potatoes, he told his protégé. A conductor beats up. A ballet dancer lifts up. We grow up and outward. "Play upward," Fleisher urges his students.

After 40 years, Fleisher's own life has turned upward as well. ●



{4} name your *poison*



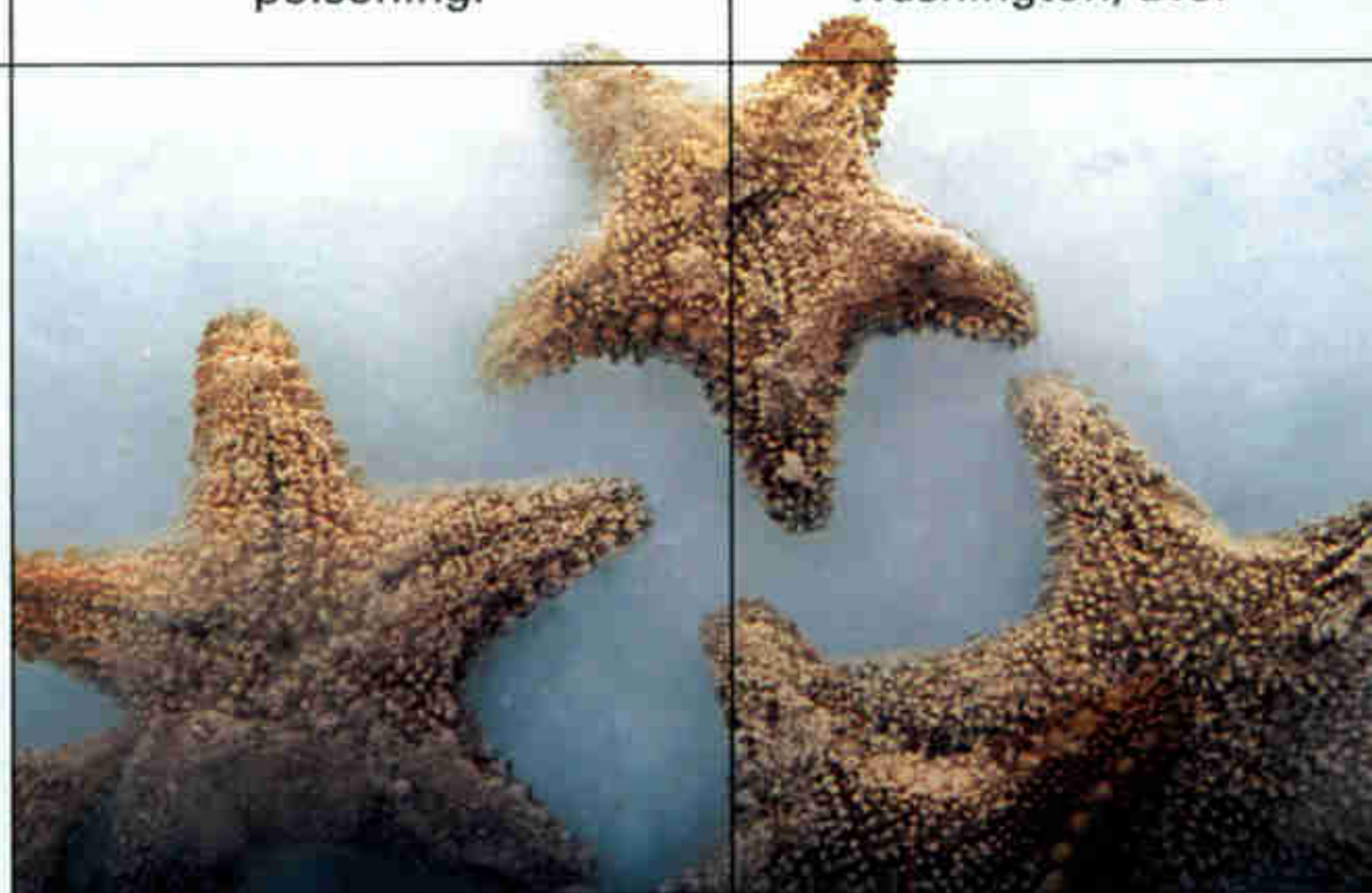
◀ Rye infected with ergot, a toxic fungus, has caused devastating epidemics through history. Symptoms include tremors and hallucinations; the hysteria of those accused of witchcraft in the 17th century may have been ergot poisoning.



▲ Spies were sometimes issued lethal pills hidden in objects like eyeglasses to use if captured. "The KGB grabbed spies by the throat so they couldn't swallow," says Peter Earnest of the International Spy Museum in Washington, D.C.

▼ A popcorn cat poisoned several New England children in 1955, when levels of orange food coloring reached toxic levels due to poor manufacturing controls. Victims recovered, and the manufacturer recalled the other cats.

► The National Cancer Institute evaluates marine-animal toxins for potential cancer drugs. Animals with no armor and limited mobility rely on poison for defense. NCI scientist David Newman calls it "animal chemical warfare."



▼ Georgi Markov, a Bulgarian dissident, was assassinated in London in 1978 when a man approached and jabbed him with an umbrella modified to fire a pellet with ricin, a deadly toxin. This replica is cut away to show the firing mechanism.



◀ In 1971 a man in Bedford, New York, died of botulinum poisoning after eating vichyssoise made by the Bon Vivant Company. Over a million cans of possibly under-processed soup were recalled. The company filed for bankruptcy.



ERGOT: IVAX; EYEGLASSES, UMBRELLA: INTERNATIONAL SPY MUSEUM, WASHINGTON, D.C.; SOUP, POPCORN CAT: HISTORY OFFICE, FDA

{5} Meet the fugu, aka *Takifugu rubripes*, a fish with the thick-lipped, thuggish face of a Chicago gangster. Fugu, or puffer fish, as it is commonly known, is a delicacy in Japan. It can also be deadly. Those who eat the liver, ovaries, gonads, intestines, or skin swallow tetrodotoxin, a powerful neurotoxin that jams the flow of sodium ions into nerve cells and stops nerve impulses dead in their tracks. They run the risk of suffering the fate of the famous Kabuki actor Mitsugoro Bando, who in 1975 spent a night feasting on fugu liver because he enjoyed the pleasant tingling it created on his tongue and lips. The tingling was followed by paralysis of his arms and legs, difficulty breathing, then, eight hours later—death. There is no known antidote.

Fortunately, these days the making of a fugu chef is a carefully controlled and licensed enterprise. Aspiring chefs who would spend their days in the kitchen skinning and shaving the fugu into tissue-thin slices for sashimi (at \$500 a plate) must take an exam: 20 minutes to dissect the fish into edible and inedible pieces, label the parts with plastic tags (red for toxic, black for edible), and prepare an artful arrangement. Of the 900 hopefuls who took last year's exam, 63 percent passed.

The source of the fugu's poison is a subject of debate. Tamao Noguchi, a researcher at Nagasaki University, believes the secret lies in the fugu's diet. Puffer fish, he explains, ingest toxins from small organisms—mollusks, worms, or shellfish—that have in turn ingested a toxic bacterium known as vibrio. In experiments, Noguchi has raised fugu in cages, controlled their diet, and produced toxin-free fish.

He hopes his research will result in the state-sanctioned sale of fugu liver. "A great delicacy; once you eat, you cannot stop," he says. Japan has forbidden the sale of fugu liver since 1983; before the ban, deaths of those who overindulged in the liver, or ate it by mistake, numbered in the hundreds.

If Noguchi succeeds in his efforts, gourmands may have cause to cheer, though the fish itself, he speculates, may have cause to mourn. "After all," he says, "a fugu without its poison is like a samurai without his sword."

Kendo Matsumura, a research biologist at the Yamaguchi Prefectural Research Institute of Public Health, discounts Noguchi's deadly diet theory. He says the fugu's toxicity comes from poison glands beneath its skin. Some fugu are poisonous, he says, some aren't, but even experts can't tell which is which.

Place your bets. Matsumura has never eaten fugu. "I am not a gambling man," he says. However, Noguchi considers it the ne plus ultra of fine dining.

When it comes to fugu, one man's *poisson* is another man's poison. •

a delicacy to *die* for



in the *morgue*



{6}

Marcella Fierro is chief medical examiner of the Commonwealth

of Virginia and a professor in the Department of Legal Medicine at Virginia Commonwealth University School of Medicine in Richmond. She oversees the medical investigation of all violent, suspicious, and unnatural deaths in Virginia, and she inspired the character Kay Scarpetta in Patricia Cornwell's crime novels. Alphonse Poklis is director of toxicology and professor of pathology, chemistry, forensics, pharmacology, and toxicology at VCU. He works with Fierro to analyze medical evidence in homicide cases and testifies as an expert in court.

When does the red flag go up? How do you know you're dealing with a murder by poison?

MF: There are a couple of presentations. If someone takes a huge overdose of something toxic, you expect a classic range of symptoms even a first-year resident can pick up on. Chronic poisonings—when toxins are fed slowly, continuously—are easier to misdiagnose. Antifreeze in the Gatorade was a recent case. A common warning sign is when the clinical history is florid. For example, lots of trips to the internist for weird symptoms or stomach pains. The victim doesn't feel well; it's diffuse, nonspecific. Of course over time classic elements of poisoning may present: He doesn't eat, he's losing weight, he's

sounding more teched each day. It looks like natural disease, but isn't.

At what point do you get called in?

MF: We see any death that is sudden, unexpected, violent, or where there is allegation of foul play. If we have the body before it's in the ground, we deal with it. But often it takes time for an allegation to be made or for someone to believe it. Perhaps a family member has a motive: there's dissension about property, inheritance, a new wife, a child not getting a fair shake. Those things set a chain of events into motion. The body has to be exhumed.

Then what? How do you proceed?

MF: I take umpteen tissue samples at autopsy: heart, liver, lungs, brain, spleen, hair, nails. Blood tells you what was going on in the body at the time of death. Vitreous humor from the eye is great. It's clean. No fermentation or contamination from bacteria. Al and I work together. What poisons are candidates? What best to collect? You have to have a strategy. We'd want to know what poison the defendant would have access to. If it's a farmer, we look for agricultural things like pesticides or herbicides. We need to have an idea of where we are going. We can easily run out of tissue and blood samples before we run out of tests to do.

So the technology you use to detect poisons in a corpse must be pretty sophisticated?

AP: Very. I call it the vanishing zero. In the 1960s it took 25

with al and marcella

milliliters of blood to detect morphine. Today we can use one milliliter to do the same work. In terms of sensitivity, we've gone from micrograms to nanograms, which is parts per billion, to parts per trillion with mass spectrometry. You can find anything if you do the research. Of course some substances are more apparent. You can smell cyanide the minute you open a body at autopsy. Cyanide works fast—like in movies where the captured spy bites on the capsule and dies. It's a chemical suffocation; cyanide hits the mitochondria in the cells, and every cell is deprived of oxygen. You die quickly, dramatically, violently.

Is there a personality profile specific to poisoners?

AP: The poisoner tries to cover up what he does, as opposed to somebody who shoots, strangles, or rapes you. A forensic psychologist I know calls poisoners custodial killers. Often you are dealing with a family situation. It happens over a period of months or a year. The perpetrator is taking care of the victim, watching him die. Poison is the weapon of controlling, sneaky people with no conscience, no sorrow, no remorse. They are scary, manipulative; if you weren't convinced by the evidence, you wouldn't believe they could do such a thing.

MF: Al sees the poisoner as a controller. I see the poisoner as a smooth psychopath who could lie to Christ on the Cross, and you would believe him. I only know of two who pled guilty.

A case that sticks in your mind?

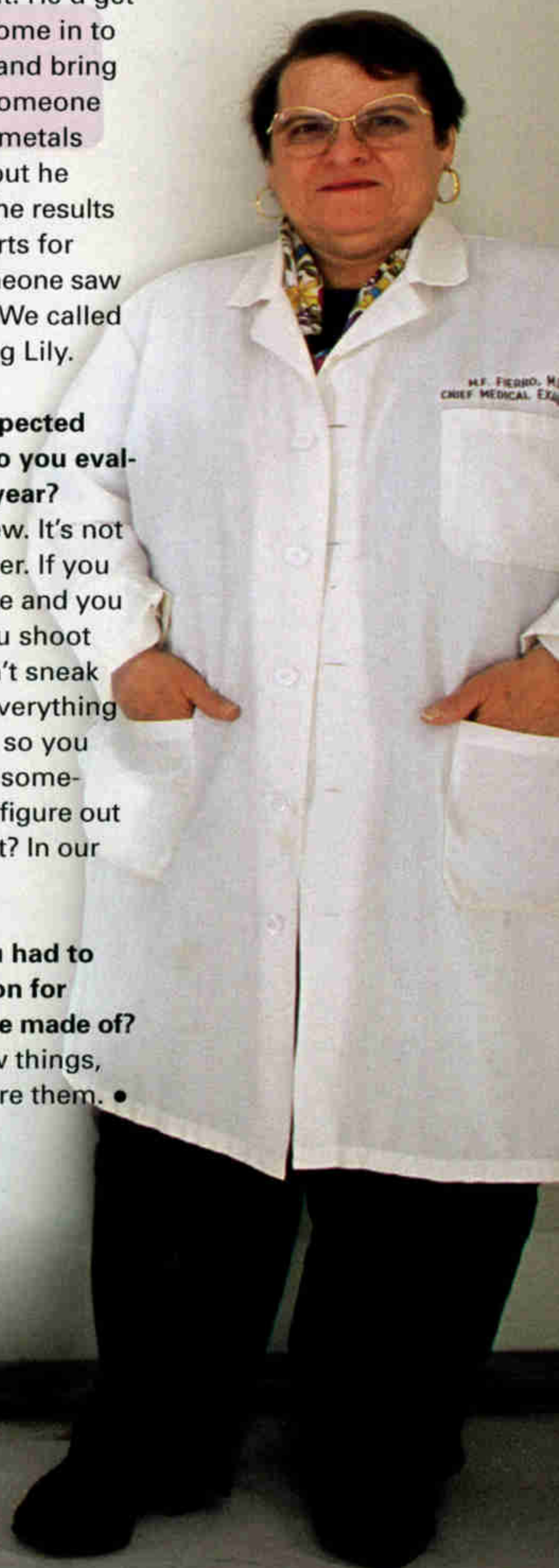
MF: There was this fellow at the University of Virginia hospital. Kept getting admitted for weird gastrointestinal complaints. The doctors were twisting themselves inside out to figure it out. He'd get better; his wife would come in to see him in the hospital and bring him banana pudding. Someone finally ordered a heavy metals [toxicity tests] on him, but he was discharged before the results came back—off the charts for arsenic. By the time someone saw the labs it was too late. We called the wife Banana Pudding Lily.

How many cases of suspected homicidal poisonings do you evaluate in the course of a year?

AP: Frankly, relatively few. It's not in the American character. If you are going to kill someone and you are a true American, you shoot them. A real man doesn't sneak around. In our culture everything is solved in 30 minutes, so you aren't going to plan, go someplace to get poison, and figure out How am I going to give it? In our culture, we act directly.

You're the expert. If you had to design the perfect poison for murder, what would it be made of?

AP: I could think of a few things, but I'm not going to share them. •



{7} When you think about it, not much has changed in 500 years. Spies, assassinations, covert contracts, secret payoffs—it's all part of the everyday business of running a country.

In Renaissance Italy "poison was the solution to delicate political problems," says Paolo Preto, a professor of modern history at the University of Padua. So it should be no surprise that poisoning was as much an art as painting, architecture, or sculpture. A touch of arsenic, hemlock, or hellebore added to the wine was discreet, nearly undetectable (autopsies were rare at the time), and considerably less messy than using a knife or gun.

The Borgias—Alexander VI and his son Cesare—specialized in faith-based poisonings. As pope, Alexander appointed wealthy men as bishops and cardinals, allowed them to increase their holdings, then invited them to dinner. The house wine, dry, with overtones of arsenic, neatly dispatched the guests, whose wealth, by church law, then reverted to their host. English essayist Max Beerbohm wrote: "The Borgias selected and laid down rare poisons in their cellars with as much thought as they gave to their vintage wines. Though you would often in the 15th century have heard the snobbish Roman say . . . 'I am dining with the Borgias tonight,' no Roman ever was able to say 'I dined last night with the Borgias.'"

But the capital of conspiracy in Italy was Venice, where the architects of evil were the Council of Ten, a special tribunal created to avert plots and crimes against the state. To accomplish poisoning, the council would contract with an assassin, usually from another city. The deed, when done, was paid for through an intermediary. Funds were readily available for such matters, and the council kept two accountings: one for public dealings and one for those of a private nature.

The council's cloak-and-poison-dagger proceedings were recorded officially (opposite, bottom) in a thin volume marked *Segreto Secretissima* ("top top secret"). Those present swore twice on the Bible to keep the meetings secret, forbidden even to admit they took place. Today the ledger sits in a soaring arched space in the state archives in Venice.

Consider the scheme proposed in its pages

by a doctor to a Venetian general fighting against the Turks in Dalmatia. He offered to cut the infected glands off bubonic plague victims and create a toxic potion to be spread on woolen caps, which could then be sold cheaply behind enemy lines to the Turks. Presumably, plague and buyer's remorse would result. The plot was enthusiastically endorsed by the general until someone gently reminded him that because so many Venetian troops

Dead

were stationed behind the lines in Dalmatia, his soldiers could be infected too and perish along with the enemy.

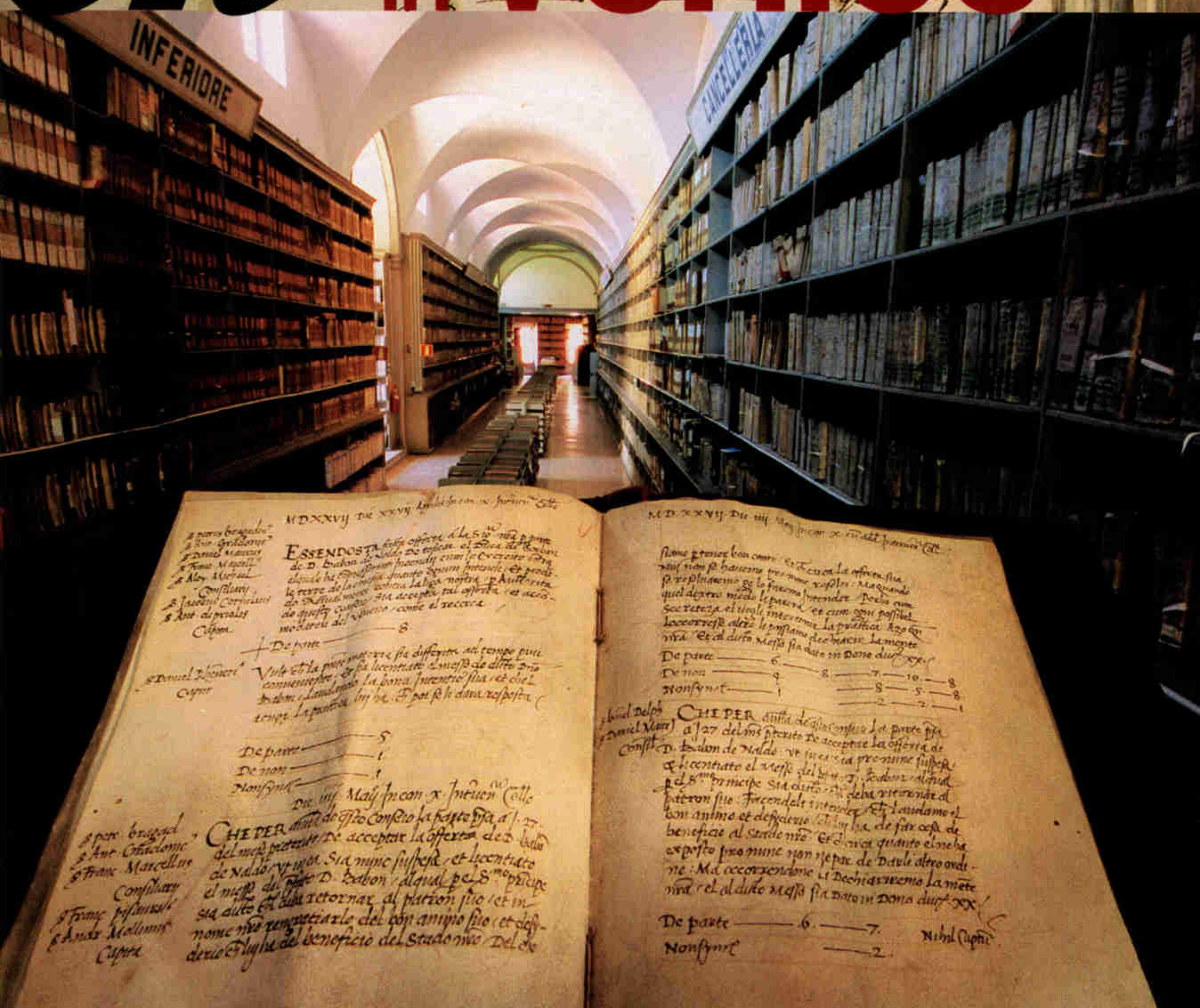
Last year poison, dioxin to be exact, was the lead player in the drama of Ukrainian President Viktor Yushchenko, victim of an attempt to remove him from the political scene. In the United States such covert plots became the subject of congressional investigations after the early 1960s, when the elimination of Cuban dictator Fidel Castro was a top CIA priority. Mobsters enlisted in the planning advised against a hail of machine-gun fire in favor of a more subtle approach: a bottle of botulinum-laced pills. Other plans, considered then rejected, included the delivery of a box of botulinum-soaked cigars, contaminating Castro's scuba breathing apparatus with tubercle bacilli, or sprinkling his shoes with thallium salts in hopes that hair loss, one of the common side effects of thallium absorption, would make his beard fall off.

Though the recurring narrative of poisoning plots might lead one to despair for the human race, Paolo Preto, who spent eight years researching dark dealings in the Venetian state, takes a pragmatic approach. "History is made up of bad acts," he says. ●

Spinta a Venetia il 24 d'agosto
della M^{te} sig^{ra} chiara Caterina
era moglie del M^{te} sig^{ro} V.
S^{er}mo donno morto & ha
mangiato di uelena e sopra
cui ha fatto V. M^{te} 25
in c^{ia} nella Villa di S. Lazzaro

Maria fia de S Batt piattar de
postquallin Kate Marto Subia S Marchetta

in Venice





zyklon b

and the camp of death



*In the summer of 1941 Himmler informed me of the following:
"The Fuhrer has ordered the final solution of the Jewish question.
We, the SS, are to carry out the order. The existing extermination sites
in the east cannot cope with the large scale of the planned operation.
I have therefore designated Auschwitz for this purpose."*

—Rudolf Höss, Commandant, Auschwitz

{8} On September 3, 1941, at Auschwitz, a concentration camp in Poland, Nazi security guards forced 600 Soviet prisoners of war and 250 ill inmates into a locked room. They poured pellets of Zyklon B, a crystallized form of hydrogen cyanide normally used as an insecticide, through a vent and watched.

Previous mass killings had been carried out by shooting squads or by pumping exhaust fumes into sealed vans. The former method, however, was too slow and created too much of a public spectacle; the latter was unreliable and required special equipment.

The Zyklon B pellets proved effective, efficient, and infallible. Exposed to air, they turned to gas, which killed all occupants of the room in 20 minutes. After the experiment the Nazis built four larger, permanent gas chambers and crematories in Birkenau, a sub-camp of Auschwitz. The key to the final solution, Adolf Hitler's plan to exterminate the Jews of Europe, was Zyklon B.

Stefan Polchlopek (left), who grew up and still lives in Krynica, Poland, was arrested by the Gestapo on December 28, 1942. He was 26 years old, a law-school graduate, and an active member of the resistance. When he was arrested, someone told his mother, who ran to the railway tracks and managed to wave goodbye to her son as he was hauled away.

Polchlopek was taken to a collection point, then put on another train for Birkenau. The car was a solid, stifling mass of prisoners. When the

train stopped, he recalls, "the doors opened; we heard shots, howling of dogs, and screams. Searchlights glared in our faces. They told us to jump off, and we fell to an indescribable hell."

In the summer of 1943, Polchlopek worked in a labor crew assigned to extend the railway line from a depot outside the camp right up to the gas chambers. Transports from throughout Europe were arriving two or three a day. Jews, Gypsies, political dissidents like Polchlopek, homosexuals—anyone considered undesirable by the Nazis—were unloaded

from railcars and either taken to the gas chambers or consigned to slave labor.

One day, an SS officer approached Polchlopek and three other prisoners working on the line and ordered them into the undressing room, the chamber in front of the room where the gassings took place. He made them collect the clothes and belongings of those who had been killed.

"I saw the undressing room and the gas chamber," Polchlopek, now 89, says. "I remember the showerheads. I remember the

stings & arrows

{9} History is one long arms race—from sticks and stones to nuclear weapons.

According to Adrienne Mayor, a classical folklorist, the Greek superhero Hercules invented the first biological weapon described in Western literature, and it's been downhill ever since.

Hercules slew Hydra, a mythical many-headed serpent, then dipped his arrows in the venom to ensure their lethality. The legacy endures in the word "toxic," from *toxikon*, Greek for poison arrow.

In A.D. 199 the Romans attacked Hatra, a city in today's Iraq. Citizens retaliated by lobbing clay pots filled with deadly scorpions (re-creation, right) over the walls. Hannibal had devised a similar strategy 400 years earlier. His sailors catapulted pots full of venomous snakes onto the decks of the opposing fleet. In Neolithic times, some scholars suggest, a plugged beehive tossed into a cave may have flushed an enemy out.

Other biological weapons in history's armory of terror include the smallpox-infected blankets the British sent to American Indians during the French and Indian Wars; the animal carcasses thrown by Confederate forces into wells during the U.S. Civil War; the sharp bamboo stakes smeared with feces by the Vietcong.

Today's toxic weaponry includes anthrax

letters, which killed five people in the U.S.

in 2001, and sarin, which killed 12 when members of a cult released the poison gas in a Tokyo subway in 1995. Such grim reality prompts defensive maneuvers like the exercise held by the U.S. Capitol Police last November in Washington, D.C.—a rehearsal for a scenario in which a poisonous substance is released in the Capitol Building.

What goes around comes around. Along with enemies, Hercules' poison arrows killed old friends and innocent bystanders. Ultimately, the law of unintended consequences claimed Hercules too. Tricked by one of his victims, Hercules made the fatal mistake of putting on a robe dipped in Hydra venom. The mythmakers specialized in irony.

Before Hercules died, he passed his poison arrows on to Philoctetes, a gifted archer, who killed many soldiers in the Trojan War. Death begets death, but—at least this time—reason prevailed. Philoctetes decided not to pass his deadly arrows on to a younger generation. He founded a temple and left the poison arrows behind. In a gesture of hope, he dedicated them to Apollo, god of healing. •

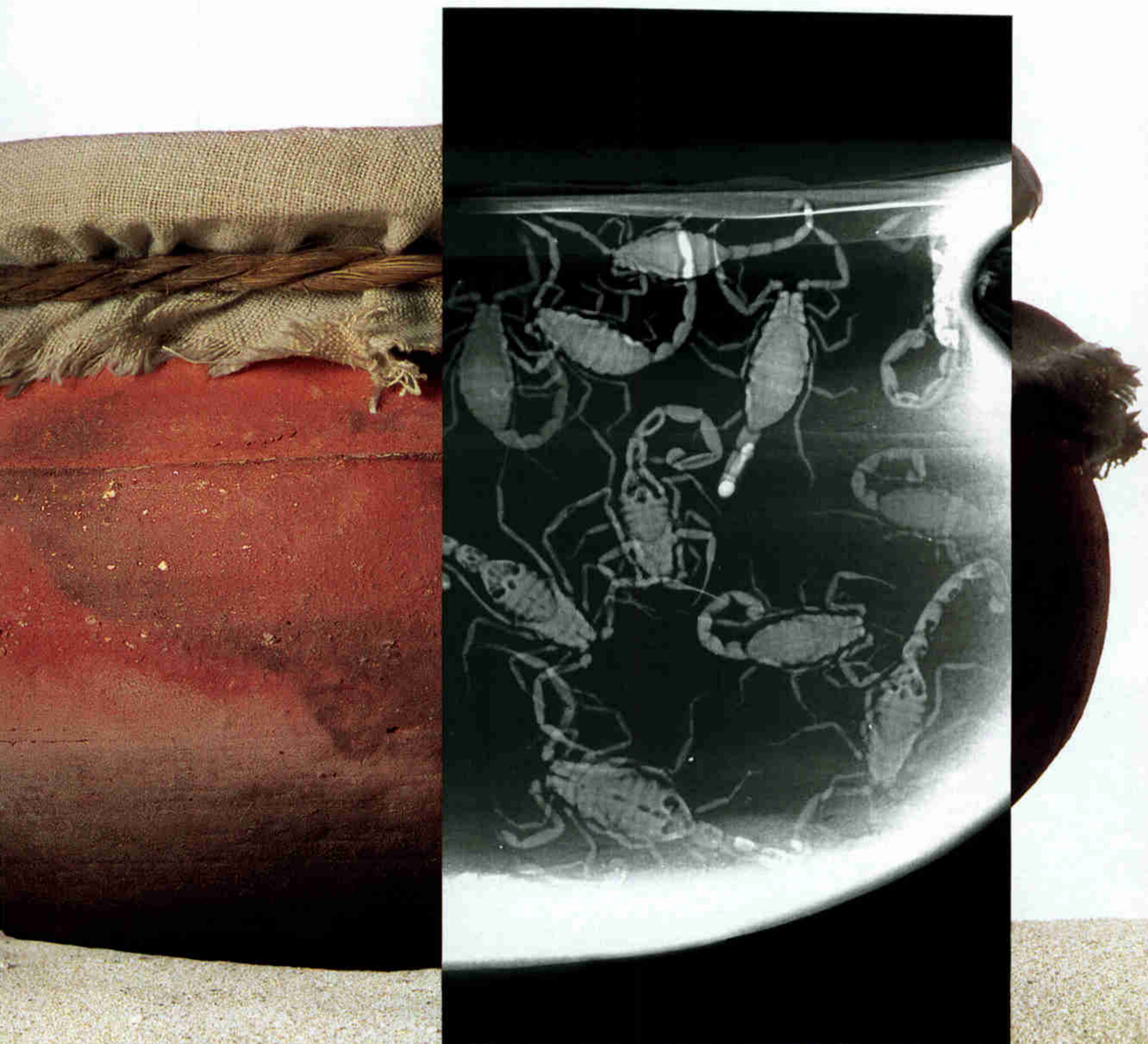


clothes, shoes, the personal possessions left in pockets. We had to gather up the clothes and load them into trucks. The belongings would go to warehouses where they would be sorted. The smell of burned corpses was in the air; dark smoke poured out of the chimneys. We realized we should flee. Witnesses were killed. We could be next." And so they fled. They ran back to the barracks.

"Everyone knew about the chambers. Once I saw two trucks crowded with women. They knew where they were going. One woman

was praying. One was cursing. All were screaming. They were followed by two trucks filled with firewood. The women were killed with Zyklon B. The naked corpses were taken out, thrown into pits, and burned."

At the height of operations, nearly 8,000 people were gassed each day at Auschwitz-Birkenau. By November 1944, more than one million men, women, and children had died. "Those of us who survived Birkenau are assured a place in heaven," Polchlopek says. "We have already experienced hell." ●



the monk who

{10} To live according to the precepts of a stringent religion can be difficult. To die by the precepts of a religion is another thing altogether.

In the shadow of Mount Yudono in Japan's Yamagata prefecture, the landscape lifts into a corrugated carpet of evergreen. This is the land of mummified priests, those who have, in a purification rite known as the "thousand day training," deliberately poisoned—and at the same time preserved—their bodies in compliance with the teachings of a ninth-century monk named Kukai, follower of an esoteric sect of Buddhism called Shingon.

"It is the principle of 'I suffer so that you might live,'" explains Yugaku Endo, the chief priest (95th in a line) of the Dainichibo temple, home to one of 27 such mummified priests in Japan.

For 76 years, Yugaku Endo recounts, the priest known as Daijuku Bosatsu Shinnyokai Shonin lived in austerity. He ate nothing except berries, bark, and nuts. He spent his days and nights climbing in the mountains, through the heat of summer and snows of winter.

Finally he sensed his days were coming to an end and ate nothing. He fasted on the idea of starvation and self-sacrifice. He grew thin, then thinner. He sipped tea

made from the toxic sap of the urushi tree, used to make lacquer. Near the end he drank only from hot-spring waters that, unbeknownst to him, contained high levels of arsenic.

The urushi sap, a purgative, induced vomiting and

urination, desiccating the priest's body. Arsenic, a preservative, killed bacteria that would cause decay. Shriveling, emaciated, he withered away. When he died in 1783 at 96, he was buried in a mound of earth and stones. Three years later, when exhumed, his skin looked as if lacquered onto a skeleton. He had become a *sokushinbutsu*, instant Buddha.

Often we die as we have lived. The brave die bravely. Cowards die cowardly.



embalmed himself

Through history poison has served such ends. Socrates, sentenced to death by an Athenian jury in 399 B.C., on charges of corrupting the city's youth and interfering with its religion, accepted the judgment with grace,

drank hemlock, and died in the company of his friends. Defiant Cleopatra, preferring death to being paraded as a spoil of war by the conquering Roman Octavian, opted, it is said, for the fatal bite of an asp. Adolf Hitler, confronted with defeat, chose cyanide (after first dosing his Alsatian to test the toxin's efficiency).

Today Daijuku Bosatsu Shinnyokai Shonin resides in a glass case in the Dainichibo temple, wrapped in red and gold robes. He sits in a pose of meditation—a man of holy belief shriveled

by time, the tradition of his religion, and the deliberate ingestion of poison, intent on serving others through the suffering and obliteration of the self. •



along came a

{11} Chuck Kristensen has 70,000 mouths to feed and didn't get to bed until 6 a.m., so he is entitled to doze off in the middle of an interview. Kristensen's dependents are spiders: 20,000 black widow babies, thousands of brown recluses and tarantulas, and a few scorpion species besides. The horde constitutes the holdings of Kristensen's company, SpiderPharm. It takes 16 hours to get the spider cafeteria in order each day. No sooner is one meal finished then it's time for the next. The round-the-clock menu includes four sizes of houseflies and fruit flies, wax worms, and, for the tarantulas, an occasional mouse.

Kristensen raises spiders for their venom, which he extracts into tiny vials. It is powerful stuff. A black widow bite can cause severe pain and muscle spasms in a recipient. Brown recluse venom degrades tissue and produces a gangrene-like wound. Funnel spider venom leads to trembling, increased blood pressure, and vomiting. Other spider venoms punch holes in cell membranes, leading to cell death.

Kristensen sends his vials of spider venom to scientists around the world because poison, the death dealer, teaches about life as well. Roderick MacKinnon, winner of the 2003 Nobel Prize in chemistry, used tarantula and scorpion venom to help decipher the structure and function of potassium ion channels in cells.

Ion channels are conduits, like gates, that control the transmission of electrical impulses within cells. Because their opening and shutting in the cell's membrane controls the entry of potassium, calcium, sodium, or chloride ions, the channels and their receptors act as on-off switches that allow a thought, a heartbeat, a breath, the lift of an eyebrow to proceed—or not.

Tarantula toxins can stimulate receptors to hold a gate open in the neurological equivalent of an electrical surge, or slam it shut in the equivalent of a power failure. A busted gate provokes conditions ranging from numbing to outright paralysis on one end to muscle contractions or convulsions on the other. The same malfunction can provoke high blood pressure, cardiac arrhythmia, or epilepsy.

Spider venoms provoke such potent physiological responses that they turn a spider into a virtual Svengali. But why doesn't a spider just knock out its prey and sit down to lunch? In life things are always complicated, Kristensen says. A tree spider may not want a fast knockout: Its meal would curl up and fall out of the tree. Paralysis is the better option. It's the insect equivalent of the surgical strike.

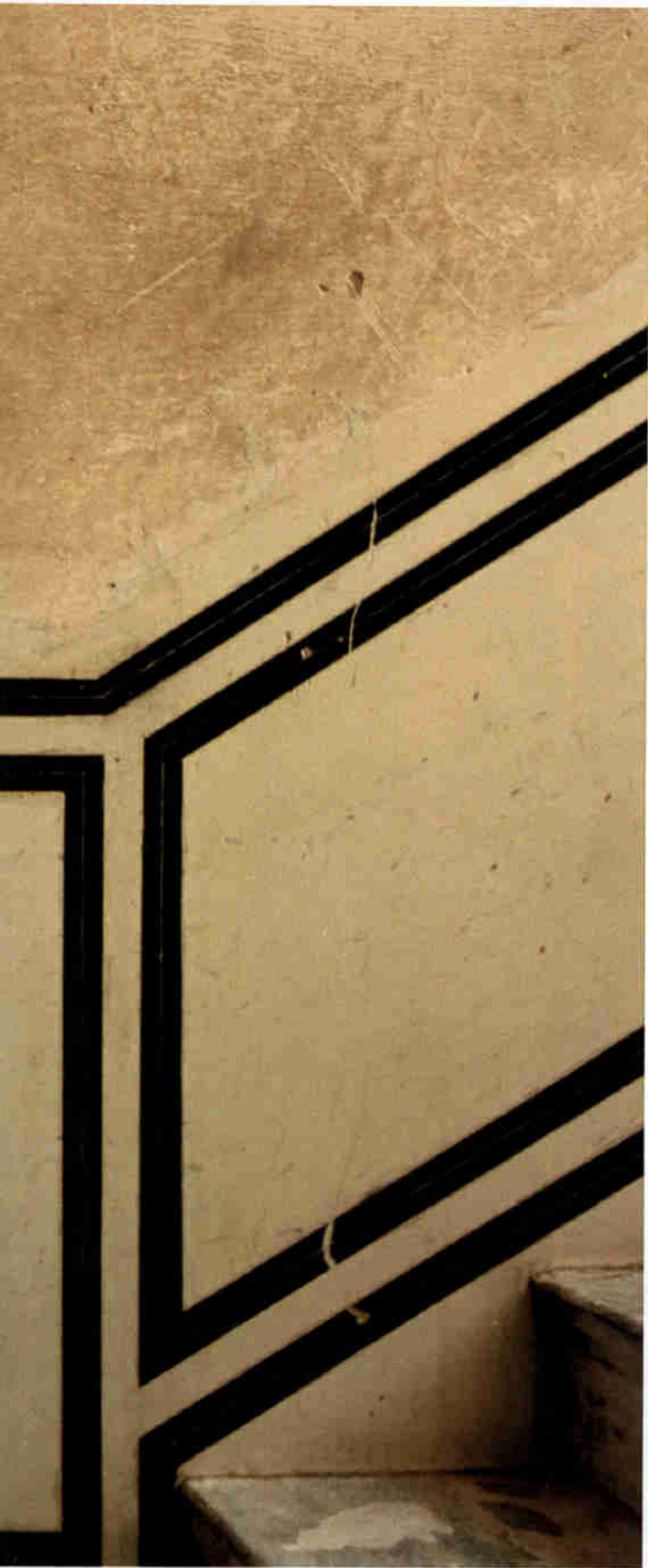
So scientists seek the chemical mastery of the spider. Says Kristensen, "Who controls potassium channels controls the world." •



spider...



when your first bite might be your
last



{12} Among the occupational hazards of being king, tsar, or maharaja, few are so permanently incapacitating as a pinch of arsenic slipped into the soup. For that the royals have long had a remedy: the food taster.

For three generations the family of Mathura Prasad held the post of food taster to the *thakur*, or lord, of Castle Mandawa in India's Thar desert. "Food was kept under lock and key," he recalls. Before entering the kitchen, "the cook would bathe and change into different clothes. Guards would check his pockets and turban to make sure he wasn't hiding anything. Only then would he be allowed in. When the food was ready, some from each dish would be fed to a dog. Next I would taste, then the guards. The food would go to table under armed escort. Several trusted generals would test it. Finally, the lord and his guest would exchange bits of each dish. Just in case."

Such things are no longer done at Castle Mandawa, now a hotel. But recently, when the vice president of India came to lunch, a food taster sampled the spread. Just in case. . . .

Mithridates, King of Pontus and enemy of Rome, tested poison antidotes on prisoners and nibbled a mix of 54 ingredients to protect himself against poisoning. The Roman emperor Nero commandeered slaves to differentiate between edible and poisonous mushrooms. An armed guard escorted dinner to the table at the court of Louis XIV, and Columbus carried dogs on his second voyage to taste foods his crew had to eat in exchanges of goodwill with natives of newfound cultures.

Medieval rulers experimented with crystal goblets and stones reputed to detect poison on contact. But the tried-and-true means of after-supper survival was the you-go-first food taster. By tradition, food to be tested before it was served to the ruler was set on a sideboard, or credenza. The Italian word comes from the Latin

credentia, meaning "confidence."

These days, employment opportunities for tasters are in decline. In England, Buckingham Palace reports there is no formal procedure for food tasting. "The in-house help are fully vetted," a palace spokesman says. The Japanese emperor hasn't used a food taster in years, though President George W. Bush has used Navy mess specialists to handle the job. In the state kitchens of Thailand, humans are factored out altogether. There, in an inspired example of equal opportunity employment, the taste-test heroes of the banquet table, directed by the Ministry of Health, are a legion of white mice. □

AMERICAN LANDSCAPES

State of Rock

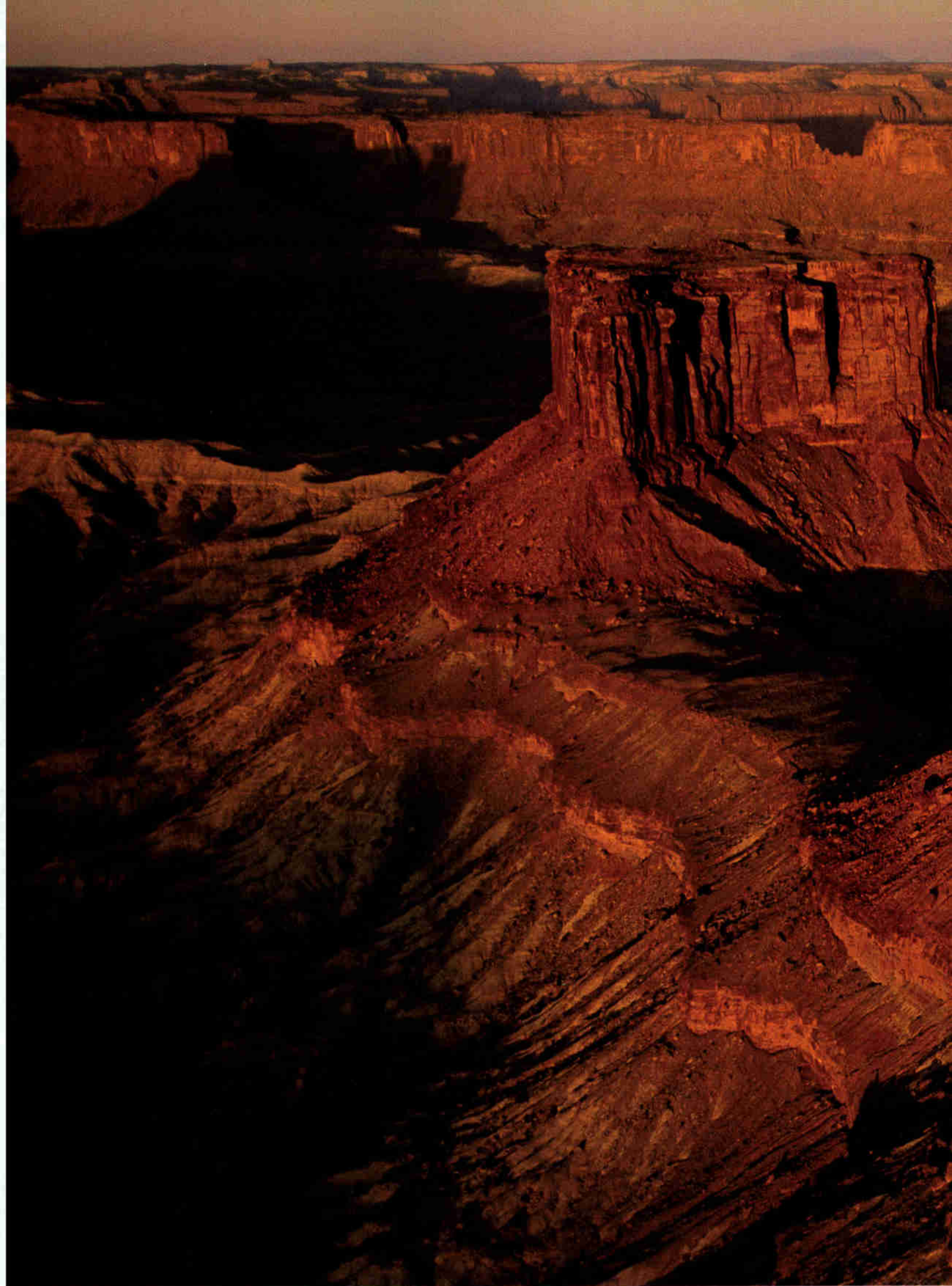


HOODOOS IN UTAH'S BRYCE CANYON NATIONAL PARK



WHERE THE ELEMENTS REIGN

Blasted by wind, broken by water, the Colorado Plateau spreads across 130,000 square miles of Arizona, New Mexico, Utah, and Colorado. This arid expanse has been called useless by some, a landscape that conspires against human settlement. For others it's nature's grandest work in progress.



ALIEN WORLDS

MELISSA FARLOW (ABOVE)



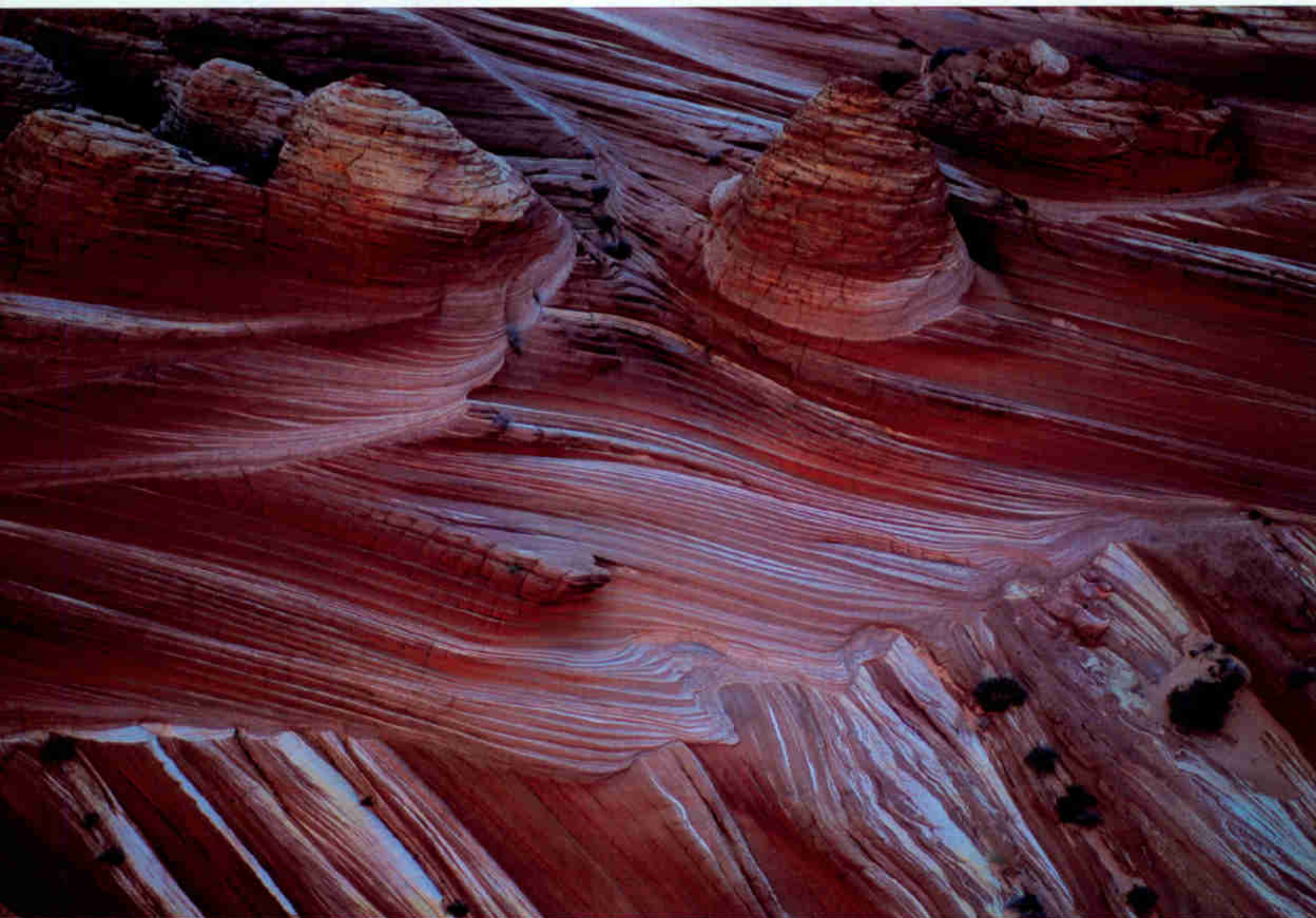
Dawn casts a Martian glow over the Buttes of the Cross in Glen Canyon National Recreation Area. Miners once scoured this backcountry for uranium. Today the mines are silent. To the west, a water-carved fist jabs across a canyon in Utah's Capitol Reef National Park (far left).



FOLLOW THE WATER

A bathtub ring of bleached rock—a sign of severe drought—lines Lake Powell, the country's second largest man-made lake and canteen for much of the Southwest. Five years of drought and demand from distant cities has depleted the lake's reserves to their lowest point since 1969. "This is the driest five years we've seen for a century," says Tom Ryan, a hydrologist with the U.S. Bureau of Reclamation, the agency that monitors the lake. Farther north, Factory Butte (right) looms over unproductive furrows of shale, salty sediments deposited by an ancient sea.





BY MIKE EDWARDS PHOTOGRAPHS BY FRANS LANTING

Bizarre. Is that the right word for the Colorado Plateau, this thirsty sprawl of gaudy-hued stone festooned with such names as Hell Roaring Canyon, Scorpion Gulch, and Horsethief Point?

Edward Abbey began his classic *Desert Solitaire* with the simple “This is the most beautiful place on earth.” Fiery rock can do that to a man. Others trying to understand the seductive pull of the plateau country apply adjectives like “amazing” and “awesome.” Which aren’t incorrect, merely inadequate. In truth, a single adjective may not suffice. All the same, as I fly over the plateau on a May morning, looking down on whalebacks of slickrock, on crashing waves of rock, on minarets and pyramids of rock hewn by water and wind—how could any word fit better than “bizarre”? Especially in Utah, the bizzarrest precinct of this Great State of Rock, which is almost as big as three Ohios and sprawls into Arizona, New Mexico, and Colorado.

Desert this is, but water’s tattoo is everywhere. Spidery little arroyos coalesce into bigger arroyos

that plunge into the still deeper groove of a river, maybe into the thousand-foot-deep canyon of the Escalante, a scalpel-cut in red rock, so narrow that the stream and its fringe of willows and tamarisks are invisible unless you’re dead-on overhead.

Most of the collected runoff, if it hasn’t vaporized or died in a mudflat, swells the Colorado River. By the time the river courses into Arizona and roars into the plateau country’s most dazzling feature, the Grand Canyon, it is plowing a furrow more than a mile deep. Pretty impressive digging, this, considering that the precipitation in parts of the plateau averages only six inches a year.

Water was also present at the creation, in far greater abundance. Tens of millions of years ago, seas, swamps, and rivers deposited dozens of layers of rock: limestones, mudstones, shales, many



LIQUID LANDSCAPES

Swelling beneath Arizona's Coyote Buttes (left), sandstone waves evoke white water. Photographer Adriel Heisey found a paradise of form as he soared over bands of shale and sandstone (above). "Everywhere I turned," he says, "there were geometric patterns that defied my ability to comprehend."

reddened by traces of iron. In those eons the plateau country was flat and much lower than its heights today, which are typically 5,000 feet above sea level. Winds also contributed raw material, the makings of sandstone layers hundreds of feet thick. The whole shebang was thrust upward by forces within the Earth. Colliding tectonic plates tilted and bent layers like cardboard. Omnipotent then as now, water attacked the soft stones, carving canyons. That's Plateau Geology 101, slightly abbreviated.

Winging 2,500 feet over this rockscape, I feel a tenuous kinship with John Wesley Powell. On his scary hundred-day journey down the Green and Colorado Rivers in 1869, that indefatigable adventurer-scientist surmounted cliff tops to reconnoiter the uncharted territory he had penetrated. Of course, I'm riding shotgun in a

Cessna while Powell had to pull himself up one-armed from a riverine chasm to a lookout; he lost his right arm at Shiloh in the Civil War. But we gaped at the same sights. "The landscape everywhere . . .," he wrote, "is of rock—cliffs of rock, tables of rock, plateaus of rock, crags of rock—ten thousand strangely carved forms."

Clearly Powell was awed by the fantastical convolutions of this land. He became the chief expositor of the "plateau province," as he called it, documenting not only the geology but also the ways and lineages of its Indians, meanwhile campaigning for sensible husbanding of the water.

Powell hadn't glimpsed some of the craziest rock shapes—the pinnacle-like hoodoos of Bryce Canyon or the vaulting spans of Arches, canonized by Edward Abbey. Bryce and Arches are two of the roughly 30 parks and other national



preserves that make the heart of the province one of the nation's most protected regions. It isn't perfect protection; environmentalists decry its insufficiencies while locals, ingrained with the Westerner's mistrust of bureaucracy, grumble about overkill. In the largest unit, the 1.9-million-acre national monument Grand Staircase-Escalante, created only in 1996, off-road vehicles plow tracks that won't disappear for decades. On the other hand many mining claims have been relinquished or bought out by those devious Feds, and no new claims are permitted. And while allowing multiple uses, including ranching (yes, there's a little grass here and there), the Bureau of Land Management is charged with superintending the monument, to protect its attributes.

Gaudy vistas are only one of those attributes. The plateau is a time machine nonpareil, holding who knows what secrets. When the rocks at the bottom of the Grand Canyon are counted in, the swath of Earth's history exposed by water's





WRINKLES IN TIME

Like human skin, the Earth's outer layers reveal the sensuous—and the severe. At its vast scale (map), the plateau's complexion is best seen from the air. Shale blankets the landscape in curves and folds (left). Elsewhere, fractures in sandstone form car-size blisters (above).

relentless gouging of the plateau is reckoned by geologists to reach back 1.7 billion years, more than a third of Earth's existence.

One afternoon, puffing along behind Alan Titus, a BLM paleontologist, I dropped back a mere 75 million years or so. Titus assured me we were tramping through a swamp where ferns and magnolias had flourished, although it looked awfully like a forest of stunted piñons and junipers. "The whistles and shrieks you hear are not birds," Titus said, coaxing my imagination into play. "They're dinosaurs." Soon he had me seeing huge crocodiles and snakes sloshing lazily in warm pools. And then, guiding me to a row of dark, roundish objects half-buried in the soil, Titus said: "You're looking at the remains of an animal that's been extinct for 65 million years or more"—the fossilized vertebrae of a

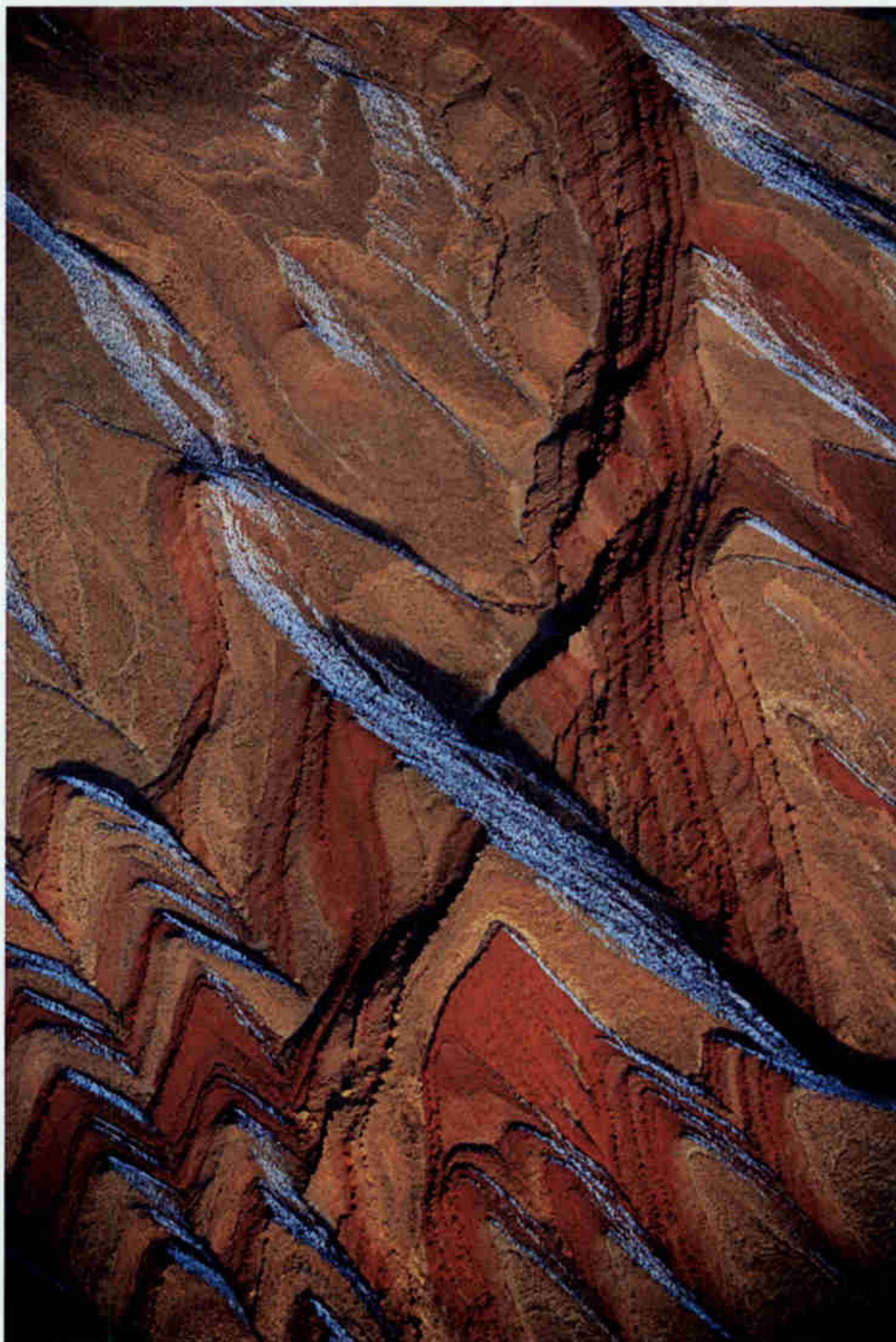
duck-billed dinosaur that lived in the Cretaceous period. Titus says this specimen was 25 feet long. "I'm waiting for that big tyrannosaur," he said. "I want to find one before I retire."

On another afternoon I climbed to a high cliff's edge. The setting sun infused the rocky layers vaulting away to the horizon with a crimson incandescence—the kind of glow, surely, that compelled Edward Abbey to pronounce these rocks beautiful. A hot, dry wind came up, gusting stronger and stronger, and as it assaulted the cliff faces it whined and screamed.

Sounded just like dinosaur shrieks.

ON THE ROCKS Sift through hypnotic images in our online gallery and reserve your own piece of the plateau by downloading one of photographer Frans Lanting's geographic gems as wallpaper at nationalgeographic.com/magazine/0505.



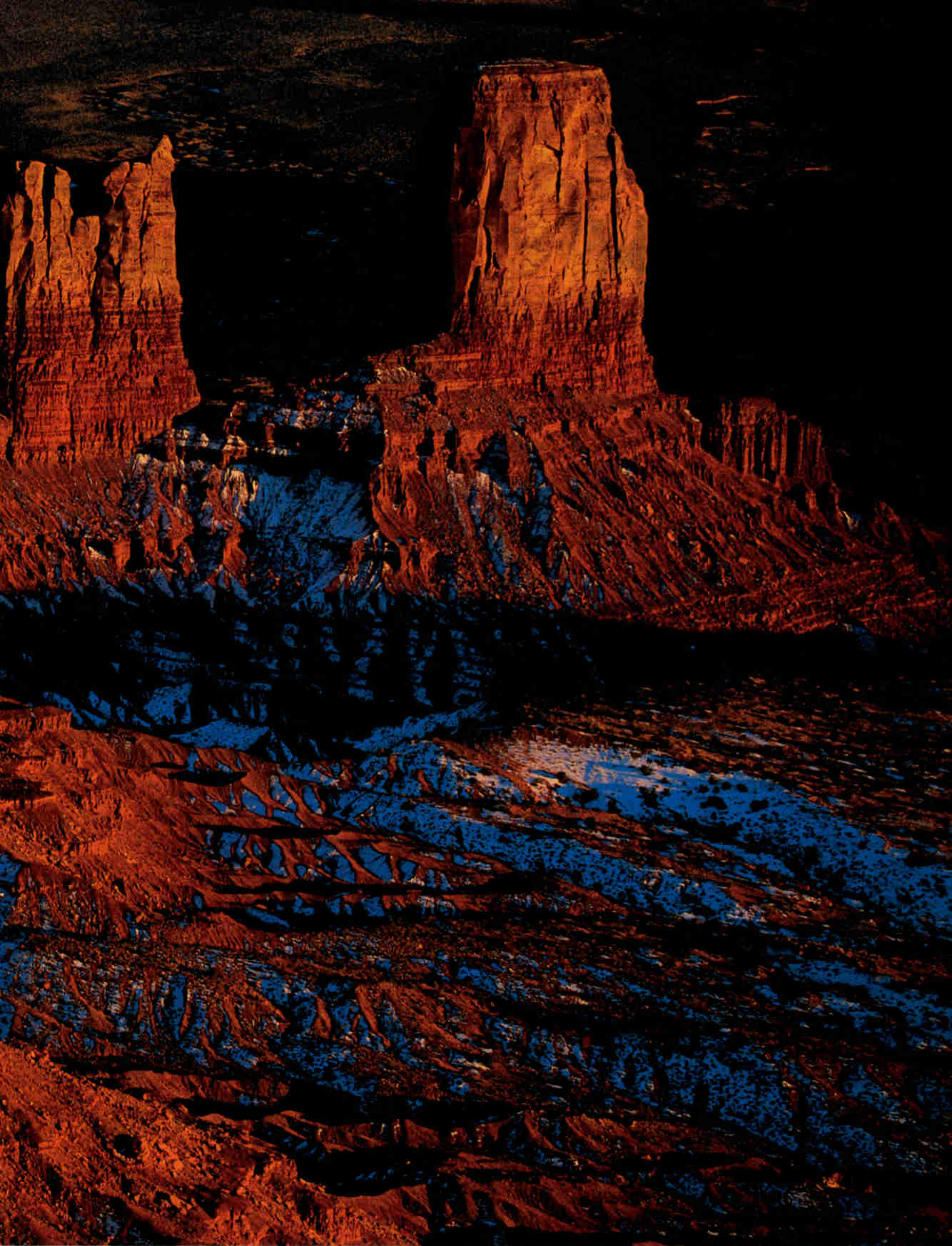


ISLANDS OF MEMORY

Echoing with legends, the plateau preserves collective memories of death, loneliness, and inspiration. Patchwork clouds yield, and sunlight pours over Jacobs Chair (left), a Utah butte named for a cattleman who drowned fording a storm-swollen creek nearby. In the late 1800s Mormon pioneers left a trail of names as they hacked a wagon route across southern Utah during the grueling Hole-in-the-Rock expedition. Near Bluff, Utah, snow powders hogbacks along Comb Ridge (above).



W I D E - O P E N C A N V A S



Standing tall against the cold, Castle Rock, at far right, and other buttes bear a dusting of snow in Monument Valley. With each season the elements shape the plateau anew, shattering canyon walls and cliff faces, chiseling subtle changes into the enduring symbols of the West. □

By **LEWIS M. SIMONS**
Photographs by **LYNN JOHNSON**

Fyodor Shidlovskiy and Mike Triebold don't know each other from a hole in the ground, but they share an undying passion for long-dead and buried beasts.

Each summer Shidlovskiy mounts a safari of men, buses, trucks, amphibious vehicles, planes, helicopters, and river-boats and ventures onto the tundra of northeastern Siberia. In the extended Arctic daylight, he and his team spend weeks at a stretch recovering the bones and tusks of woolly mammoths, the lumbering precursors of today's elephants that until about 10,000 years ago wandered the bitterly cold steppes alongside our own fur-clad ancestors.

The best of his finds he restores (with auto body filler and varnish) and assembles into complete skeletons. Bones and tusks of lesser quality he has carved into chess sets and knick-knacks. The least valuable are



FOSSIL WARS



Dispatch From the Front Lines

in Siberia, are part of a growing international trade in fossils that's as lucrative as it is controversial.





Riding high, a 21-foot model of an *Allosaurus* heads to a Manhattan auction house. "It's a hot market now," says Roby Braun of Cycad Productions, whose sculptures can fetch \$50,000 or more from museums hungry to satisfy demand for all things dinosaur—including their bones.

Just as industrial barons of an earlier time bid for Great Masters, wealthy fossil fanciers compete in New York and California auction houses for the most eye-popping specimens.

ground into powder for use in traditional Chinese cures. Eventually everything is sold, mostly in Hong Kong and the United States.

Shidlovskiy (Fyodor to his friends, and everyone quickly becomes a friend of this joyful, effusive Russian) invited photographer Lynn Johnson and me to join him on an expedition. It was to be more than your run-of-the-mill Siberian mammoth quest, he promised: A hunter had tipped him to the whereabouts of an intact baby mammoth skeleton, the rarest of the rare, and he'd love to have us come along to record what promised to be an important find. Hours before dawn on a balmy August morning, we met Fyodor outside his tony, pink-brick apartment building in Moscow and prepared to set out on his latest escapade.

Two months earlier and halfway around the globe, Mike Triebold kissed his wife, J. J., goodbye at their custom-built log house in the shadow of Colorado's Pikes Peak, hopped into a convoy of four-wheel drives, and with four of his guys headed north. They were bound for the sleepy cow town of Roundup, Montana, where I would meet them. Triebold, too, was anticipating something extremely rare—a juvenile *Tyrannosaurus rex*. Walter Stein, Triebold's field manager, had recently discovered a single rib poking out of concrete-hard sandstone on private land Triebold had leased for fossil collecting. The prospect of connecting that bone to the rest of a young *T. rex* had Triebold wired and dragging heavily on his Kools.

For Shidlovskiy and Triebold, hunting down

Using a GPS device to pinpoint his location in Utah, Craig Harmon and investigators from the Bureau of Land Management determine whether a fossil dealer did his digging on public land—a federal offense if done without a permit. Fossils found on private land in the U.S.



and digging up the extremely dead is life's great joy. Besides the thrill of discovery, old bones provide them with a comfortable livelihood. In fact—with the going price for a nicely turned out *T. rex* pegged well into the millions, and a fully articulated mammoth selling for a quarter million or more—very comfortable.

Shidlovskiy and Triebold are members of a small fraternity of freewheeling men (almost exclusively) who excavate fossils and sell them for profit. I spent months tracking commercial fossil dealers and investigating their trade, not just in Siberia and Colorado, but also in Morocco, northeastern China, Montana, and the Dakotas. I discovered that some dealers are careful collectors and honest businessmen; others are disreputable and brutish, ripping bones from national parks and other protected lands and selling them for a quick buck. Still others, particularly in developing countries such as China and Morocco, are peasants striving to ease their painful lives with whatever they can claw, quite literally, from the earth around them.

During my travels I witnessed some of the damage that unscrupulous or untrained dealers do. In northeastern China I watched pick-swinging farmers hack rock slabs containing the remains of ancient birds and fish with little more concern than they gave to plowing their fields. I saw smuggled and fake fossils sold as legitimate in the United States, which strictly prohibits the excavation and export of fossils from government-owned land without a permit, but has no law banning imports—even when they've been smuggled out of their originating country.

I also watched commercial dealers excavate fossils with exquisite care, cleaning away the detritus of eons with delicate dental-style tools and keeping finely detailed records of their discoveries. Yet academic paleontologists, more than a dozen of whom I interviewed around the world, tended to tar all dealers with the same brush—as greedy yahoos and enemies of science, a charge I came to see as undeserved.

Which is not to say that every dealer runs an aboveboard business. Much of the fossil trade

belong to the landowner, who can legally sell them. At the Tucson Gem and Mineral Show, Mark Norell (below, at right), a curator at the American Museum of Natural History, examines a crocodile fossil. "Legitimate collectors have always had a role in paleontology," he says.







By day Rick Hebdon and Tanya Hester use their motel room as a showroom for their fish fossils —just one small facet of the Tucson Gem and Mineral Show. “It’s huge,” says Rick. “People fill the convention center and every motel. Even tents. Every vacant spot in town is filled.”

is in cash, and international sales often involve bribes to customs officials and police. The clandestine nature of such transactions makes it impossible to put a dollar total on the worldwide trade, but educated guesses from dealers and scientists suggest that it runs into the tens of millions of dollars each year.

The bone market boomed in the late 1980s, when dealers from Japan, flying high on an economic bubble, started buying up some of the biggest and best U.S. fossils and installing them in new museums back home. The Japanese spree drove prices beyond the reach of most American scientists and museums, which couldn't compete at auctions without the help of lavish benefactors or corporate backers. The price run-up hit an all-time high in 1997 when the McDonald's Corporation and the Walt Disney Company, in a show of marketing genius, chipped in to help Chicago's Field Museum buy a *Tyrannosaurus rex* known as Sue for a staggering 8.36 million dollars and put it on display for the world's kids to see. The sale was a media

sensation and alerted landowners across the western U.S. to the market value of the bones buried on their property. Once seen mainly as scientific curiosities, fossils were now potentially lucrative commodities.

One result is that rural property owners increasingly are turning their backs on scientists, who depend on free access to fossil sites. (Commercial dealers, by contrast, commonly pay landowners a percentage of their profits. Mike Triebold says he's paid as much as \$76,000 to one rancher.) Relationships between scientists and farm families they've visited each summer for decades have dried up in rancor.

As with any commodity, price is driven by demand, and in today's booming market, fossils compete with fine art for the attention of the super rich. Just as industrial barons of an earlier time bid for Great Masters, wealthy fossil fanciers such as Bill Gates, Nicholas Cage, and Charlie Sheen, to name a few, compete in New York and California auction houses for the most eye-popping specimens. Such fossils often end

"I own a 13-foot-long mammoth tusk, but I want the largest one that's ever been found," says John Wood, a Baptist minister in Waco, Texas, who turned his three-car garage into a trophy room featuring leopards, a jaguar, and a lion. "I call this my silent zoo," Wood says.



up in the oceanfront great rooms of the grandest Pacific coast mansions.

Paleontologists lament that once specimens disappear into private collections, they're lost to scientific study. But dealers are quick to point out that most of the world's great museums are full of important fossils donated by collectors. Indeed, many museum collections were purchased from profit-minded men (and the occasional notable woman) who went out to the Wild West of the United States, the barren wastelands of Siberia, the burning deserts of Mongolia, or the mild-mannered forests and coastlines of their own countries, and gathered the bones, teeth, and horns they found there.

Edward Drinker Cope and Othniel Charles Marsh, America's two leading paleontologists in the latter nineteenth century, both paid dealers to find the vast array of fossils they used to wage their furious "bone wars" for primacy in the field. Half a century earlier Mary Anning, a little-educated Englishwoman who grew up in a destitute family beside the coastal cliffs of Lyme

Regis, collected spectacular Jurassic fossils that she sold to scientists and noblemen throughout Europe. After she died in 1847, she was lauded as "the greatest fossilist the world ever knew."

Today's scientists say less flattering things about fossil dealers. But the opinion of scientists was far from Fyodor Shidlovskiy's mind as he bundled us aboard a chartered bus parked alongside his home. The driver ground the gears into place, and we rolled off on the first leg of an 8,000-mile haul by road, air, and water. Shidlovskiy's blue eyes, magnified behind wire-rimmed glasses, glittered with excitement.

Among us were Shidlovskiy's good friend, the Reverend John Wood, a loquacious, big-game-hunting Baptist preacher from Waco, Texas, and Wood's prominent physician neighbor, "Doctor Joe" Cunningham. They were there mainly for the adventure. (A few months later they would ship crates of medical supplies from Waco to a poorly supplied Siberian clinic we visited.)

Working beside a sunscreen at a quarry in Wyoming, Yolanda Siber digs up dinosaur bones for her family's museum in Switzerland. Private collectors and academics have a rocky relationship, says Yolanda's father, Kirby. "It's a pity. If we worked together, we'd have great results."







Bone Zone: Siberia

How do you turn old bones into hard cash? Fyodor Shidlovskiy does it working as a middleman between folks who find fossils in remote corners of Russia and collectors worldwide willing to pay top dollar for a good-looking tusk. Arriving in the Siberian village of Andryushkino, Shidlovskiy is greeted by children on sleds brimming with bones and tusks (above left), which he pays for with cash, snowmobiles, boats, and outboard motors. On another day his men ferry a load of fossils down the Kolyma River (center), then transfer the goods to a bigger boat. Twenty minutes later law enforcement officials come aboard and accuse Shidlovskiy (above, at right) of poaching fossils from a national park. Wheeling and dealing, he promises to bring the men gifts, or even hire them. A pause . . . then smiles and handshakes all around. With his fossils—and without any fines—Shidlovskiy heads home to Moscow, where he stockpiles his inventory (left). “It’s my life, my love, my hobby,” Shidlovskiy says. “And it makes me rich.”



After a 14-hour bus drive we arrived at Yoshkar Ola's derelict air base, where Fyodor hustled us onto a banged-up Russian border patrol cargo plane. This once worthy warhorse had no seats other than a few benches, no cooling, no heating, and no toilets. Already on board, to our surprise, was a cluster of Siberians living in Moscow who'd heard that Fyodor was going to their hometown and could they hitch a ride? "Fyodor just doesn't know how to say no," Wood drawled admiringly. The good news was that our gracious host had shoved a case of vodka under the pilot's seat. Less encouraging was that the pilot and navigator enthusiastically joined in the toasting as bottle after bottle made the rounds.

Eight time zones, nearly ten flying hours, and one case of vodka later, the screaming plane shuddered to a halt on the broken airstrip at Cherskiy, a no-longer-bustling port town on the Kolyma River, in the autonomous Republic of Sakha (Yakutiya) high above the Arctic Circle. Bladders bursting, backs aching, we gingerly disembarked and fell into the bear hugs of Sergei

Zimov and his wife, Galia. The Zimovs operate a tundra-monitoring scientific station outside the nearly deserted town. Sergei, an ecologist, plays host to scientists and the occasional commercial bone collector, like Fyodor, to help withstand post-perestroika privation.

Russian paleontologists are so hard-pressed that some succumb to stealing fossils from their own museums and selling them. In 1999 a complete woolly rhinoceros skeleton, as large as an SUV, was disassembled and removed from the Yakutsk State University Mammoth Museum, where it was on display in a room adjacent to the director's office. Fyodor recovered the fossil and returned it to the museum. This act, he said, won him an official open-ended permit to collect and remove fossils in Sakha and established his self-anointed reputation as "Emperor of Siberia," which he delights in calling himself.

After a day of R & R at the Zimovs' camp, we pulled on hip boots (in summer the spongy tundra above the permafrost layer can behave treacherously like quicksand) and clambered

Her face furrowed with grief, Hraa Aitkhuya mourns the death of her husband, a Moroccan fossil hunter and father of six children who was killed when a cliff collapsed as he was digging for trilobites. Running similar risks, Said Moutaouakil and Mohamed Dahbi dig for shark teeth



In developing countries such as China and Morocco, peasants strive to ease their painful lives with whatever they can claw, quite literally, from the earth around them.

onto an orange and blue Mi-8 helicopter Fyodor had chartered. We choppered 200 miles north across broken tundra to the Arctic coastline. Our first stop was a rough wooden shack alongside a snaking stream, where two of Fyodor's men and a black-and-white dog named Nelson had spent the past two months. They had collected an array of mammoth bones and tusks, which they loaded into the rear of the helicopter. Next we took off for a spot a few miles beyond, on a towering, eroded coastal cliff.

Fyodor and one of his men, Anatoly Borischuk, slithered down the black muck of the cliff face to a narrow ledge, where Borischuk earlier had spotted an adult mammoth skull about the size of a washing machine. A crew member tossed down a rope, which Fyodor threaded

through the eye sockets, and all hands helped haul the skull up and into the chopper.

Our final stop for the day was the village of Andryushkino. As our helicopter clattered to the ground, a shouting throng flooded out of their concrete-block houses to greet us, smiles creasing their broad Asian faces. The villagers, members of the Yukaghir minority, survive by herding reindeer. Some augment their limited income by gathering fossils for Fyodor, even though taboos warn against disturbing the mammoths. He pays them in cash, snowmobiles, boats, outboard motors, and most recently, laser disco equipment for a new recreation center.

A wooden sled hauled by tractor came scraping toward us, heaped with skulls, pelvises, ribs, tibias, and femurs. The bone pile was crowned

at a phosphate mine near Khouribga. "I once thought fossils were sacred, that they shouldn't be sold like carpets or dates," says an official at Morocco's Ministry of Energy and Mines. The roughly 50,000 Moroccan poor whose lives depend on this business helped change his mind.







Keeping their feet in the family business, Hussain Bouaazama and his children hunt for fossils near their village in Morocco. "Hussain makes a good living," says Abdullah Aaronson, who markets Hussain's fossils. "His house is one of the best in his village—and he's his own boss."

with a mammoth skull and twin golden tusks tall enough to shelter an NBA star. With this booty stuffed into the helicopter, the human cargo settled onto the bones of our choice for the ride back to Cherskiy.

Late the next night we drove down to Cherskiy's dilapidated port. Stumbling over rusting beams and smashed glass, we boarded a government-owned channel-marking ship for a 480-mile trip on the Kolyma River. The hunt for the baby mammoth was finally on.

As the sun set on our second day out, we anchored near the fossil site and went ashore in powerboats. We trudged clumsily in our hip boots to a shallow mud pit. There lay the large femur and a few other bones that Fyodor's man had discovered. Alas, the tributary stream that had gurgled just feet from the burial site when he made the find, only weeks before we arrived, had dried up. Russian fossil hunters customarily use pressurized water to wash away the earth entombing mammoth bones. Now the only possible water source was a tiny pool a hundred yards distant, which meant that Fyodor would need more hose. A full day was lost as another of his men sped back to Cherskiy in a small boat. When he returned, he was carrying old canvas hoses from the town's fire department and a generator for Fyodor's portable pump.

At last the long-awaited excavation began and continued around the clock for the next 48 hours. By late afternoon of the second day, the pool was nearly dry and men strained to augment the flagging flow, scooping out shovelfuls of muck as dense as chewing gum. To everyone's frustration, they uncovered only a few more bones. The youngster either had been killed at another location and part of its remains dragged by predators to this place, or over the millennia bits and pieces had been washed away by rain and melting snow. We left, Fyodor dejected.

But not for long. A few hours after setting sail back toward Cherskiy, we dropped anchor at a three-cabin settlement festooned with drying fish like silvery pennants and guarded by ferociously barking sled dogs. The previous summer Fyodor had recruited the hunter-fishermen who live here to gather whatever bones they came across, mammoth or otherwise. Now he had to rouse one of the men, sprawled in a drunken stupor on the pebble-paved riverbank. (Alcoholism is common among the lonely hunters of

the tundra.) After a few cups of steaming, sweet tea, a wanly grinning Valeriy Petrov led us to a space beneath one of the log cabins, where he and his friends had stashed tusks and bones.

Fyodor estimated the haul at over a thousand pounds and offered the men the equivalent of \$16,000, which they snapped up. He handed over a thick stack of rubles and promised to deliver a snowmobile and an outboard motor. The bones were ferried out to our ship, and we resumed the overnight run to Cherskiy. As the boat gathered speed, Fyodor began sorting through the bones, selecting out the mammoth fossils and blithely pitching rejects into the river.

About 20 minutes later we were hailed and boarded by four men wearing Environmental Protection Committee patches on their camouflage windbreakers. When confronted with a charge of poaching fossils in Kolyma National Park, Fyodor jauntily assured the officers that their concern was misplaced. Anyway, couldn't they use a new outboard motor? Not only that, but if they would collect bones for him in the future, he'd take good care of them. The erstwhile defenders of the land thanked their new benefactor profusely, pumped his hand, hopped back into their speedboat, and departed.

Turning to his bemused American onlookers, Fyodor flung his arms up in mock horror and cocked his head to one side. "Russia!" he said.

Mike Triebold's expedition to central Montana was considerably less daunting, at least in getting to where we were going. After a restful night at the Best Value Inn on Main Street in Roundup, I joined Triebold and his crew for a jouncing, hour-long ride in a pickup over prickly rangeland, eventually passing through a barbed-wire gate. As the men unloaded a pneumatic jackhammer and other tools, Triebold walked me over to a raw scar in the brown sandstone. It had been dug from the side of a jagged mound as high as a two-story house. Barely poking out of the rock was what could have been a bone in a standing rib roast, festively wrapped in aluminum foil for protection. Triebold said it belonged to a youthful dinosaur and was "float," meaning it wasn't connected to any other part of the buried skeleton.

That lone bone was the latest of a collection Triebold and his crew had recovered since they



MARK LEONG

Underground Goods: China

Want to buy or sell fossils in Liaoxi, China? The man to see is Mr. Y, here offering fish and plant fossils. Though China prohibits individuals from collecting and selling most fossils, local rules often contradict national laws. This legal morass and lax enforcement help create a money pot that lures dealers like Mr. Y—and endangers some of the world's richest fossil sites.





MARK LEONG (ALL)



Cash & Carry

The laws governing China's fossil trade may be contradictory and confusing, but people easily grasp the meaning of money. Everything is for sale at the Lingyuan Paleontological Fossil Museum, where a group of well-heeled customers eyeball the merchandise (left)—searching, they say, for “a gift for a friend.” They buy a small dinosaur fossil with a stack of bills, then disappear. Such strong demand keeps suppliers hopping. Near Chaoyang, children shoot toy guns on the shoulder of a road strip-mined for fossils (above left). At the Lingyuan Prehistoric Fossil Protectorate, poachers prowl with impunity, littering the ground with fossil shards (center). Picking through a farmer's stash of fossils in a village in Inner Mongolia, Mr. Y (above, at right) looks for something he can peddle.

“Private collecting is a headache for scientists,” says Xu Xing, a paleontologist based in Beijing. “When we try to dig or collect, local people think we're taking their treasure and try to stop us. To improve the commercial value of fossils, people doctor them, and we lose scientific information.”

“This work has to be done by experts,” says paleontologist Kevin Padian, “and an expert is someone who understands the scientific value, not the dollar value.”

began digging at the site the previous summer. Triebold, who’s been chasing fossils for more than 20 years, had reason to believe that the rest of the skeleton was buried in the mound. That meant that he and the crew would have to remove as much as ten feet of overlay before reaching the level where the animal might be. The digging commenced: the roar of a gasoline generator and the staccato outbursts of the big hammer interspersed with long periods of nothing noisier than small chisels scratching at the sandstone. Great black thunderheads boiled above us and wind-whipped sand lashed our eyes. By the time the thunderheads had damped down the dust and temperatures dipped in the late afternoon, Triebold’s crew had seemingly made no progress. Shoving back his floppy hat,

he decided to call it quits for the day. I was disappointed. He seemed not to be.

“The ground is the hardest I’ve ever worked in,” he said in a flat, midwestern twang, “but I’ve got a good feeling about this.”

Six months later, after Triebold had turned up a good portion of the skeleton, he sent me an e-mail:

“Very exciting news: One of the rib fragments has clear [*T. rex*] serrations across it. . . . We believe that this is the first direct evidence of rex-on-rex violence. Now, the big questions are these: Did a pack of Nanos [*Nanotyrannus*] kill and start eating the juvenile rex, then get chased away by a scavenging adult *T. rex*, which finished off the carcass? Or did the adult *T. rex* kill and eat the juvenile in an act of cannibalism, leaving



only the scraps for the Nanos after the adult T. rex had its fill? Or did the juvenile rex get killed by the adult rex in a territory battle, and did the carcass then get eaten by the pack of Nanos? We will be looking for more clues as we prepare the specimen.”

Along with cleaning and stabilizing the skeleton, Triebold carefully mapped the site and preserved all collateral fossils. This level of detailed data by a commercial dealer was a key factor in a decision by the Carnegie Museum of Natural History in Pittsburgh to buy a first-of-its-kind oviraptorosaur from Triebold and Fred Nuss, the fossil hunter who discovered it. The provenance was excellent, and scientists would have access to the site where the specimens were found. Carnegie is not alone: Other U.S. museums are turning to dealers for fossils.

Carnegie’s deal with Triebold infuriated some paleontologists. “We wouldn’t do it,” says Kevin Padian, curator of paleontology at the University of California, Berkeley. A dead-serious scientist with a deceptively pixieish face and

a mop of curly gray hair, Padian charges that dealers don’t take the necessary pains in establishing fossils’ taphonomy—the placement in the ground and the effects of the surrounding soil on the fossilization process. “This work has to be done by experts, and an expert is someone who understands the scientific value, not the dollar value.”


At the heart of the commerce-versus-science battle is how vast or how limited the Earth’s supply of fossils may be. “No one who thinks of conservation can think in terms of a resource being endless,” says Padian. Triebold’s response: “There’s certainly no shortage of invertebrates; they’re practically inexhaustible. And vertebrates? Even T. rexes aren’t unique anymore. Simply put, fossils are not rare.”

In a back room tour of the American Museum of Natural History’s paleontology collection, department chair Mark Norell walks me through row after row of floor-to-ceiling shelves stacked with fossils seemingly beyond count, many still in their plaster “field jackets.” “Most are awaiting preparation and have not yet been studied,” Norell acknowledges. “This is common in pretty much every museum in the world.”

While it’s true that many fossils are abundant, others are unique. Or as James Kirkland of the Utah Geological Survey put it to me, “Not all fossils are created equal. Some are worth scientific study, but many are not.”

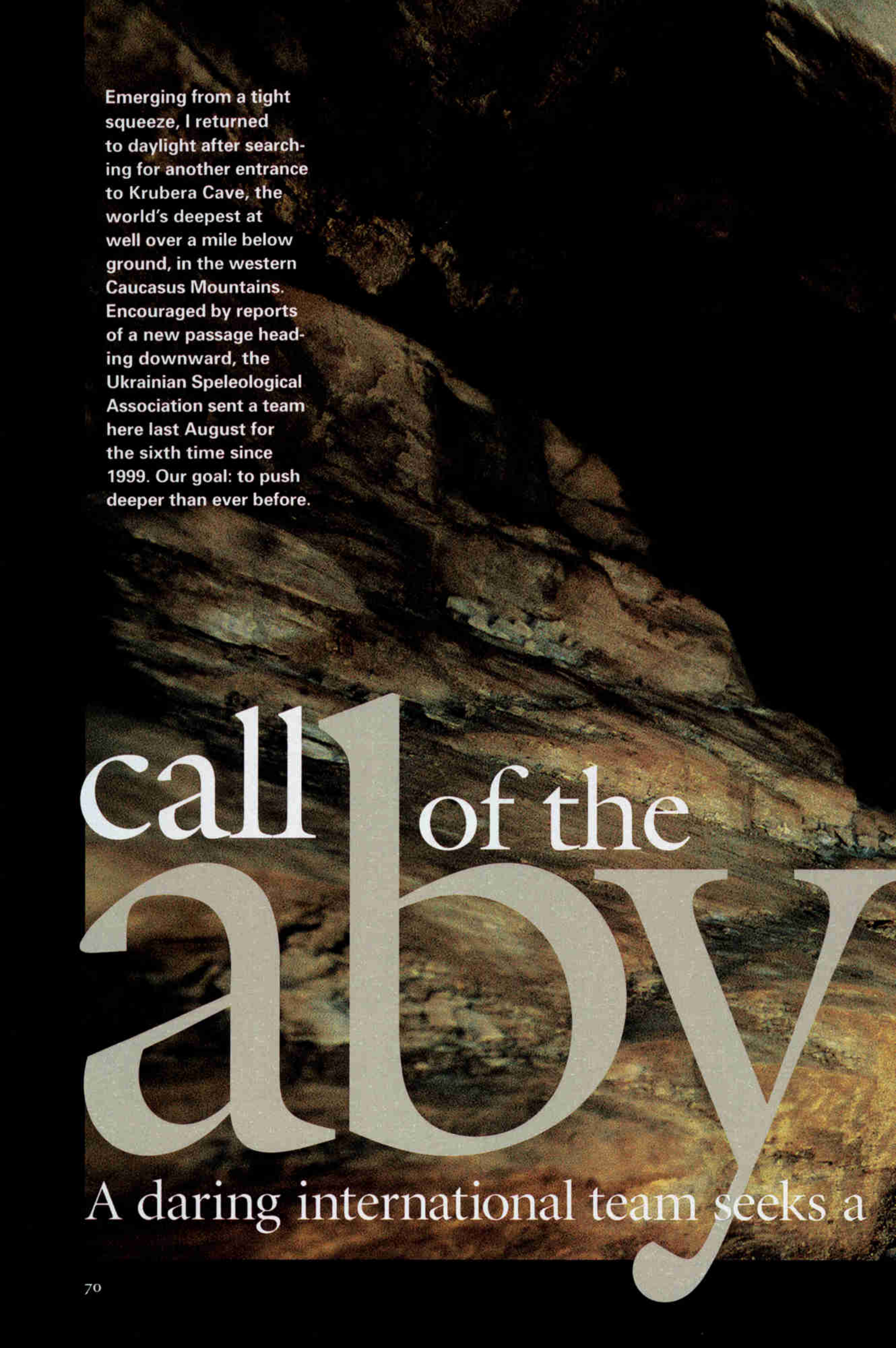
One possible solution, Padian says, may lie in dealers and academics exploring together. The scientists could keep the originals for study while the commercial people would produce high quality casts, which most museums and private collectors consider more than adequate. This would give science access to a broadened range of specimens and make material legally collected from government lands available to the private market in the form of casts.

Still, having spent many days with the two sets of stubborn protagonists, I suspect it will be a very long time before they seek each other out. Just how long might it take? We may all become fossils first. □



Earning his spurs, three-year-old Bridger Kimber puts a dinosaur through its paces in Eden, Wyoming. “You’d think the size of such creatures would frighten kids,” says Bridger’s dad, “but they’re just drawn in.” So too are hordes of fossil enthusiasts, lured by the mysteries of extinction, the thrill of the hunt, and the urge to own a piece of pre-history—or at least make a buck off it. “We’re a consumer society,” says paleontologist Laurie Bryant. “We’re used to buying whatever we want.”

RUBLES UNDER THE TABLE In a video interview, photographer Lynn Johnson talks about the shady side of the fossil trade, while author Lewis Simons examines the perspective of a poor Moroccan. Learn more, plus share your thoughts on our online forum, at nationalgeographic.com/magazine/0505.



Emerging from a tight squeeze, I returned to daylight after searching for another entrance to Krubera Cave, the world's deepest at well over a mile below ground, in the western Caucasus Mountains. Encouraged by reports of a new passage heading downward, the Ukrainian Speleological Association sent a team here last August for the sixth time since 1999. Our goal: to push deeper than ever before.

call of the abyss

A daring international team seeks a



SS

new record in the world's deepest cave

By Alexander Klimchouk
Photographs by Stephen L. Alvarez



When Sergio García-Dils de la Vega kissed his girlfriend, Pilar Orche, goodbye at the entrance to Krubera Cave, he promised to return the next day. But after teammate Bernard Tourte (above, at right) bruised his side in a tight passage, García-Dils decided to stay with him at an underground camp, missing his chance to return to the surface before going deeper. It was two weeks before Orche saw her boyfriend again.

Our expedition, however, had come prepared for a long siege, bringing more than five tons of gear to the cave. Ever since 1956, when explorers in France first descended below 1,000 meters (3,281 feet),

SOCIETY GRANT

This Expeditions Council project was supported by your Society membership.

generations of cavers had dreamed of achieving the 2,000-meter mark. Would Krubera take us there?

The underground world,
the “eighth continent,”
is one of the
last great pieces of
unfinished exploration.





Threading a giant needle, Alan Cressler rappelled into the first pit below the cave entrance. Our team rigged nearly two miles of new rope and strung a telephone wire to the bottom to keep everyone in touch with our base camp on the surface.

1 SEE MAP, PAGE 76, FOR LOCATION OF THIS PHOTO



Cutting a jagged path through the limestone of the Arabika massif on the edge of the Black Sea, the “trail” into Krubera Cave drops down a chain of pitches, cascades, and pits—some more than 100 meters deep—connected by narrow rift passages called meanders. The cave, located in the separatist region of Abkhazia, was named after Russian geologist Alexander Kruber. In 1960 researchers from the Republic of Georgia explored it to a depth of 90 meters (295 feet). Two decades later, I organized a series of expeditions to investigate new deep caves, using dye traces in cave streams to probe Arabika’s potential depth. In 2001 a team led by Ukrainian Yuri Kasjan set a world record in the cave of 1,710 meters (5,610 feet). Last July a Moscow-based team extended that to 1,775 meters. Our hope was to find a path past 2,000 meters.

At the start of the expedition, Alexander Karpechenko (top right), whose nickname is “Brick,” exulted in getting his hands on a brand new gasoline-powered hammer drill that he planned to use to bore holes for explosives to free up tight passages. Team members in nearby Snow Cave (right) cleared blasted rubble from a passage that had been blocked by a “boulder choke.”

Like mountaineers scaling a Himalaya peak, our expedition of 56 cavers from seven countries established a series of campsites, at depths of 700, 1,215, 1,410, and 1,640 meters. There team members cooked meals, slept five and six to a tent, huddled for warmth, and worked for up to 20 hours at a stretch.

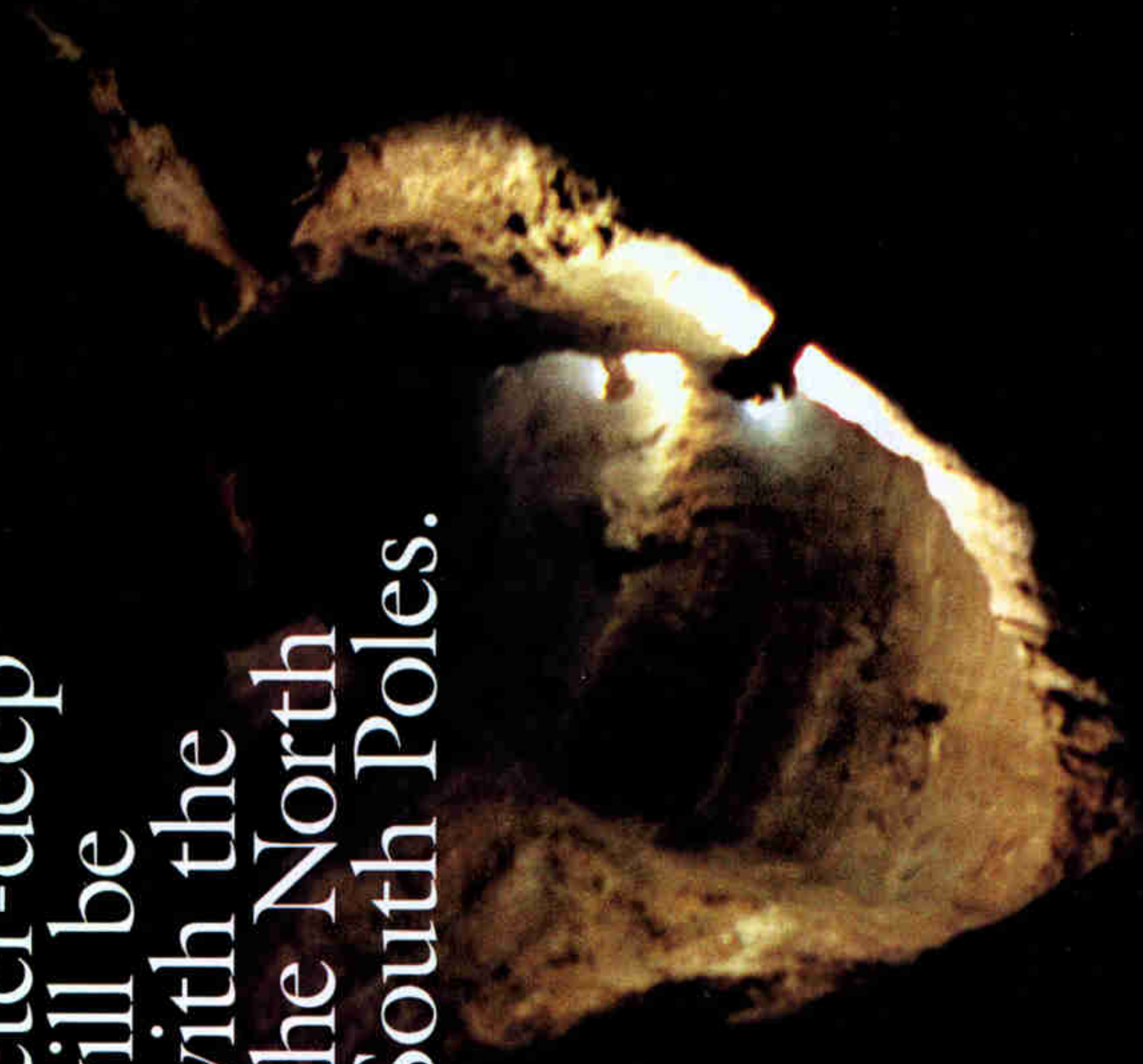
LATEST NEWS Follow the progress of explorers as they return to Krubera Cave—and check out an interactive map and more photos at nationalgeographic.com/magazine/0505.

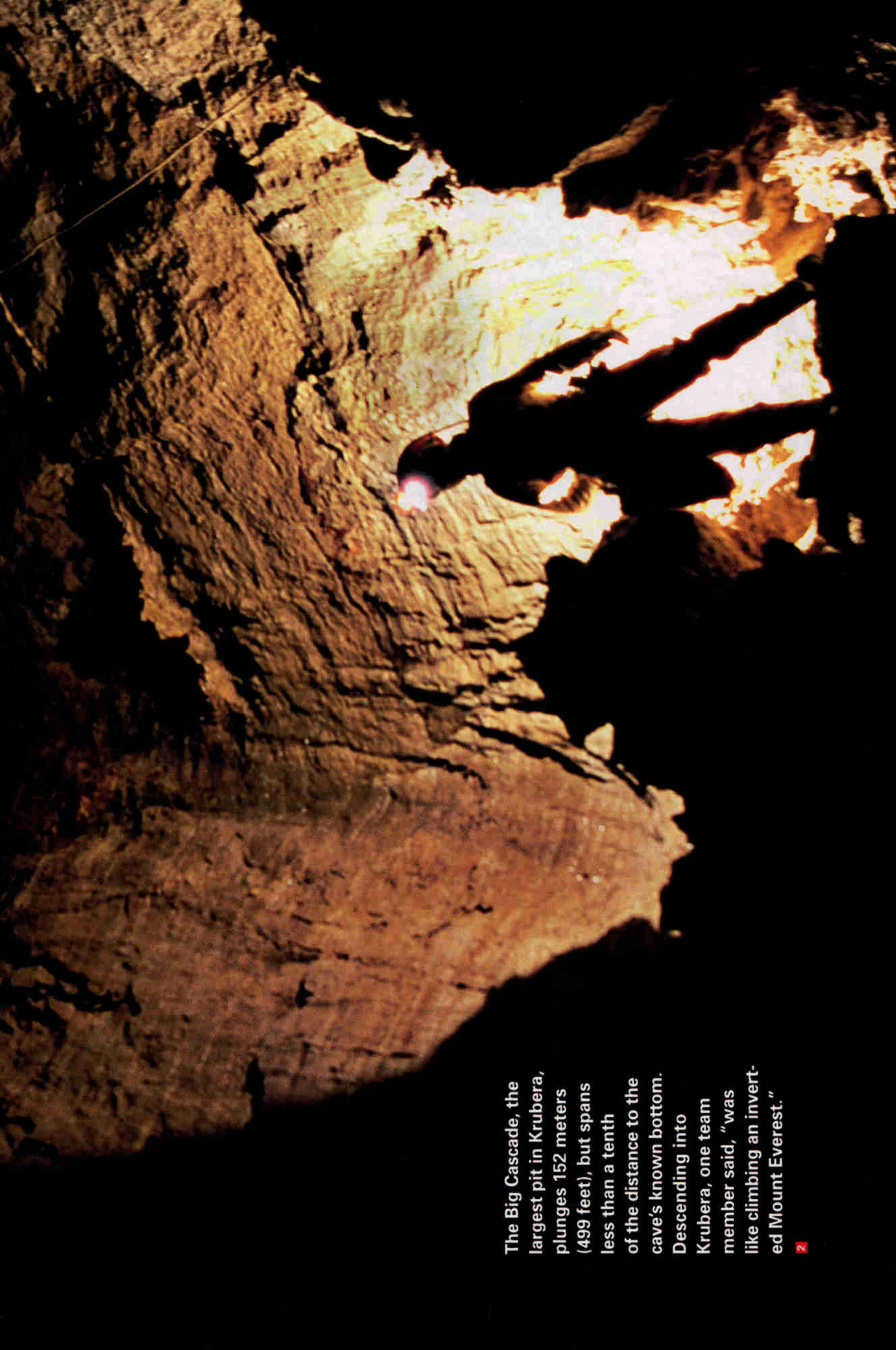


We brought more than five tons of equipment—and an enormous faith that we would succeed.



Exploration of the
first 2,000-meter-deep
cave will be
compared with the
conquest of the North
and South Poles.





The Big Cascade, the largest pit in Krubera, plunges 152 meters (499 feet), but spans less than a tenth of the distance to the cave's known bottom. Descending into Krubera, one team member said, "was like climbing an inverted Mount Everest."



By the third week our downward progress was blocked by a sump at a depth of 1,775 meters (5,823 feet). Gennadiy Samokhin (left) surfaced after a dive to examine a tight squeeze at the bottom of the ten-meter-deep pool. “No chance to get through,” he said.

Searching for a route around the sump, Sergio García-Dils de la Vega (right) braved a cascade of near-freezing water. Also unsuccessful, he discovered to his dismay that his waterproof dry suit had holes in it. “The water was so cold I lost the feeling in my fingers,” he said later. In a last-ditch effort, Denis Kurta and Dmitry Fedotov squeezed through a narrow, 100-meter-long passage called the Way to the Dream, which successfully bypassed the sump and pointed steeply down. The next day Bernard Tourte (below) and others followed. It was the breakthrough we’d hoped for. The news, spread by telephone to all camps, was greeted with elation, boosting everyone’s spirits.



BERNARD TOURTE (ABOVE AND LEFT); SERGIO GARCÍA-DILS DE LA VEGA

Water, the caver’s nemesis,
brought hazards, worries,
and numbing cold.





The newly discovered passage led to yet another sump at a record 1,840 meters (6,037 feet), where Samokhin (left) emerged smiling from a brief test dive. There was a promising downward tunnel, he reported. But it would have to wait. After nearly four weeks of working underground, with supplies running low, our expedition had finally run out of time.

Returning to the surface, with all its colors and smells, is every caver's greatest joy.

Flush with our success and relieved that team members had incurred only minor injuries, I opened my arms to welcome each of the cavers as he or she emerged back on the surface. Bernard Tourte (right), his red caving suit, helmet, and gear completely covered with grime (below), held a congratulatory bouquet of alpine flowers—for a job well done.





Four weeks later...

We'd barely returned home before a new team from our Call of the Abyss project set out to surpass the record we'd just established. In early October a group of nine Ukrainian cavers led by Yuri Kasjan went back to the trough-shaped valley above Krubera Cave (right), where a farmer's horse gamboled across the stony ground. Taking advantage of the ropes and anchors we'd left behind, they began 17 days underground. Three cavers—Ilja Lapa, Emil Vash, and Igor Ishchenko—posed with their gear bags on the way down (above left). Probing a series of “windows” in the walls of the





deepest part of the cave, the team was blocked time and again by sumps or impossibly tight squeezes. But finally, on October 19, Kasjan dropped down a pit later dubbed Millennium and looked at his altimeter. He had passed the magic 2,000-meter depth, a fact later confirmed by surveying. More pits and passages brought the explorers to a sandy chamber at 2,080 meters, the new “bottom of the world.” They named this spot Game Over. But the caving game is far from over. It won’t be—not as long as deeper abysses call out to be explored. □

SEA STARS



CLASS OPHIUROIDEA



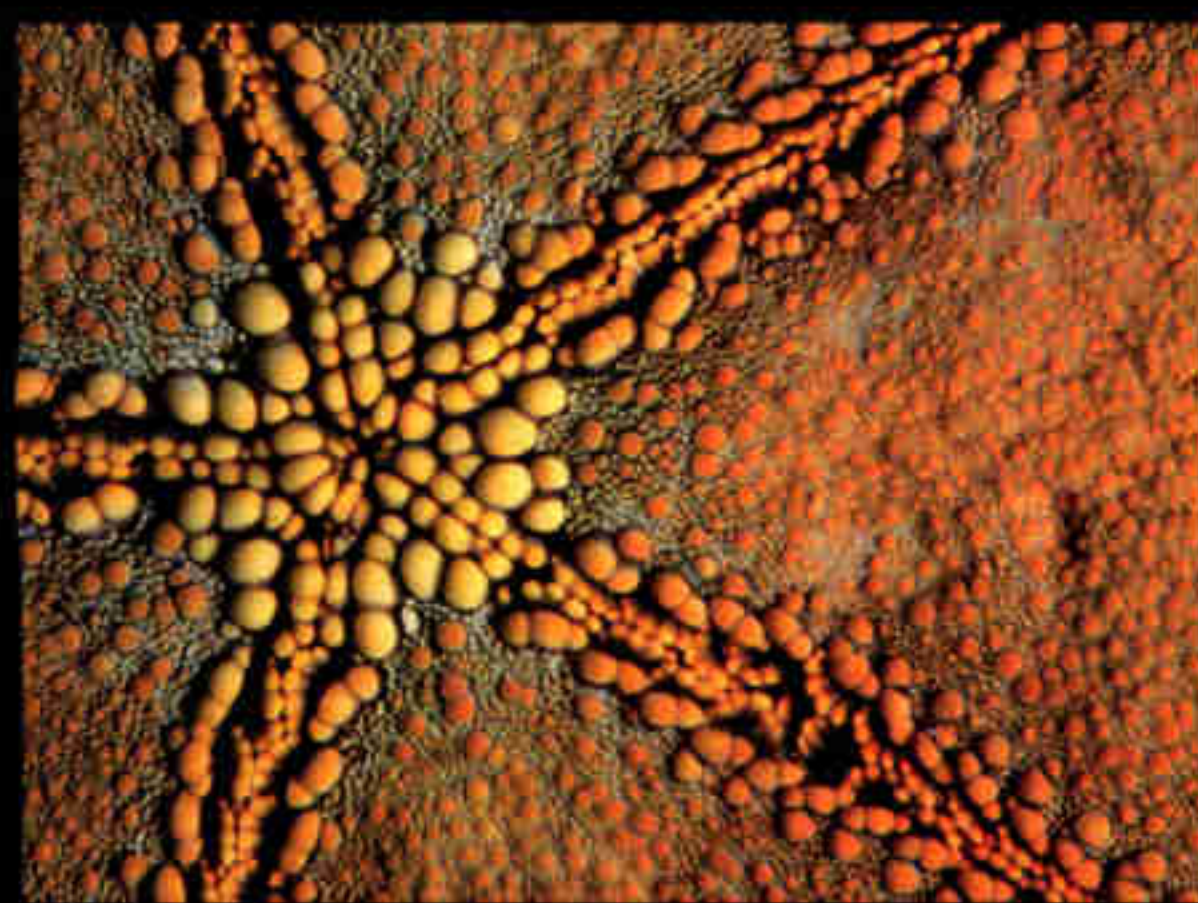
FROMIA SP.



FROMIA SP.

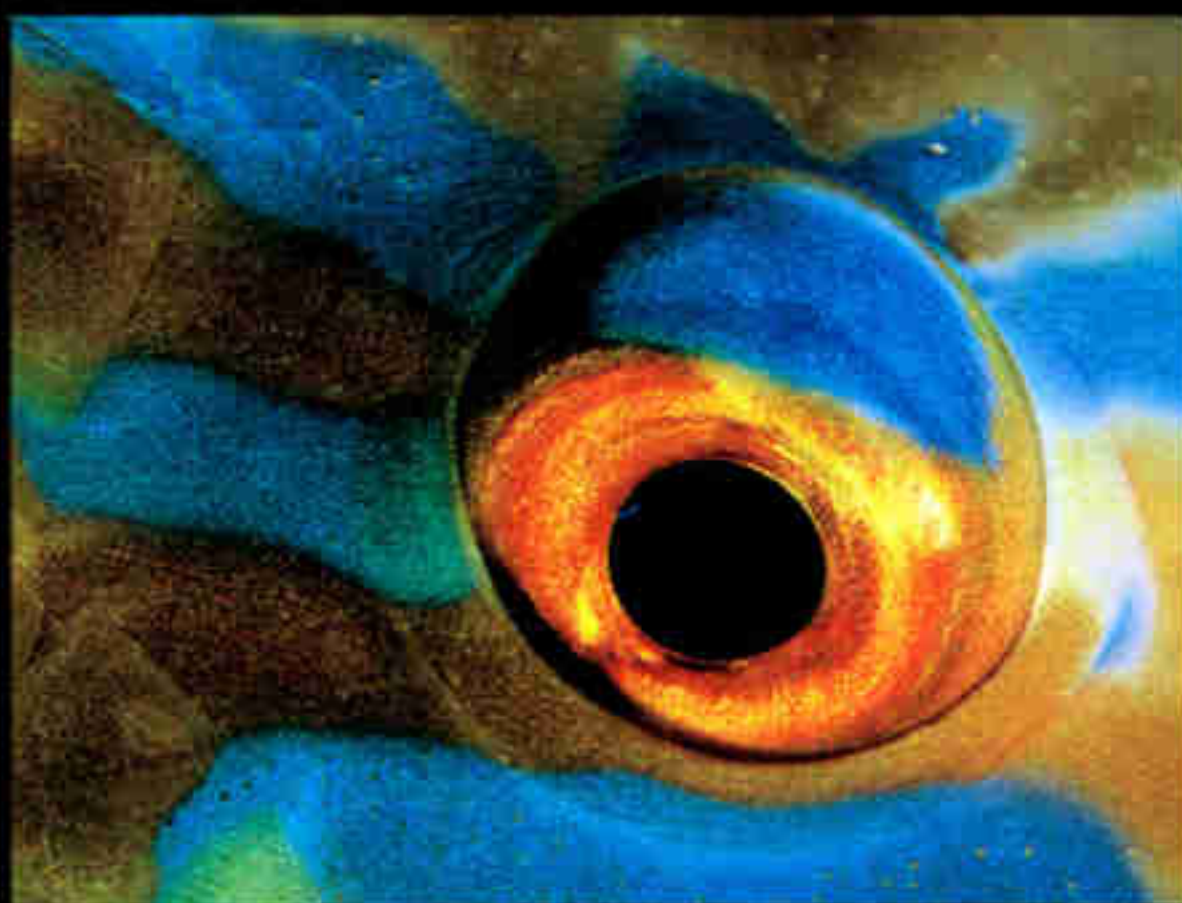


NARDOA ROSEA



CULCITA SP.

PARROTFISH



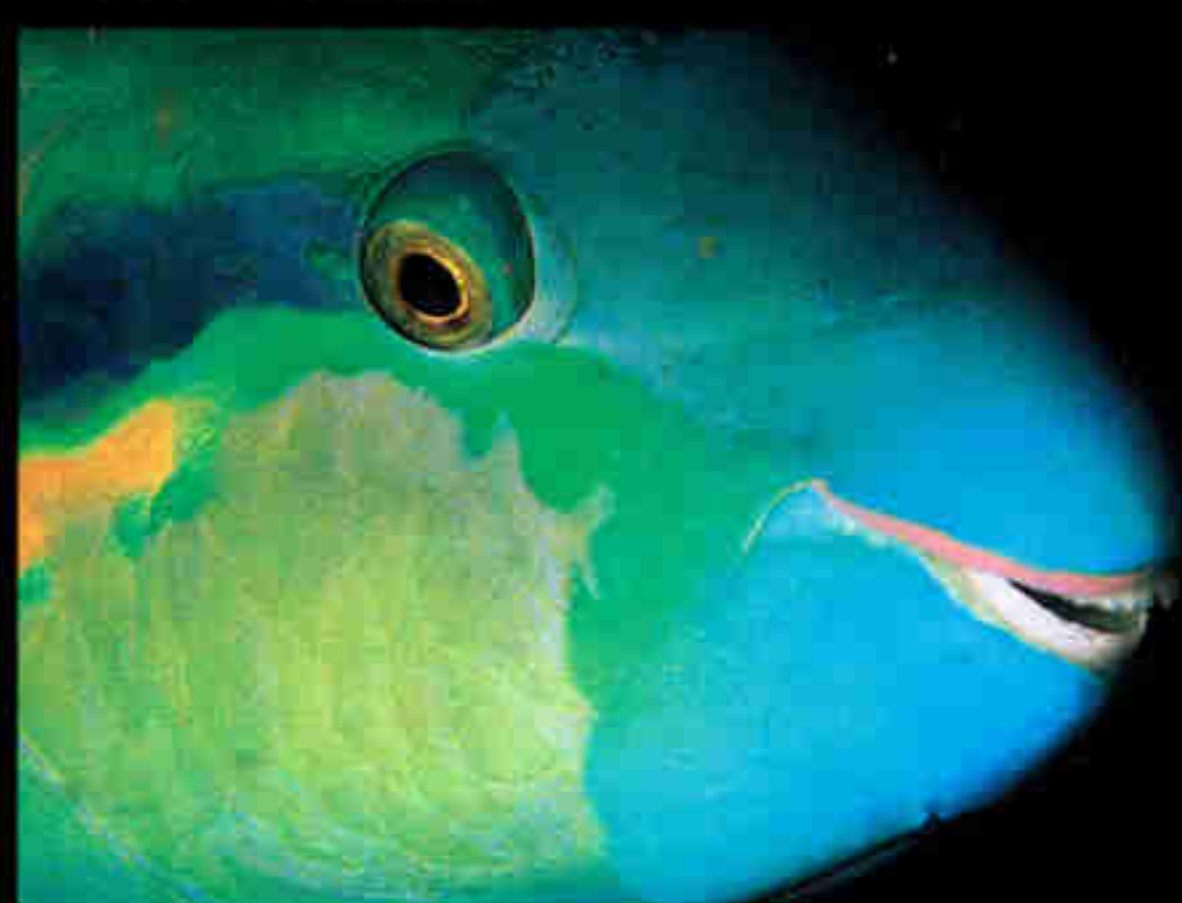
SCARUS GHOBBAN



SCARUS PRASIOGNATHOS



CETOSCARUS BICOLOR



SCARUS DIMIDIATUS



CHLORURUS BLEEKERI

one
fish
two
fish
red
fish
blue
fish
why
are
coral
reefs so
colorful?

TUNICATES



CLAVELINA SP.



FAMILY DIDEMNIDAE



CLAVELINA ROBUSTA



POLYCARPA AURATA

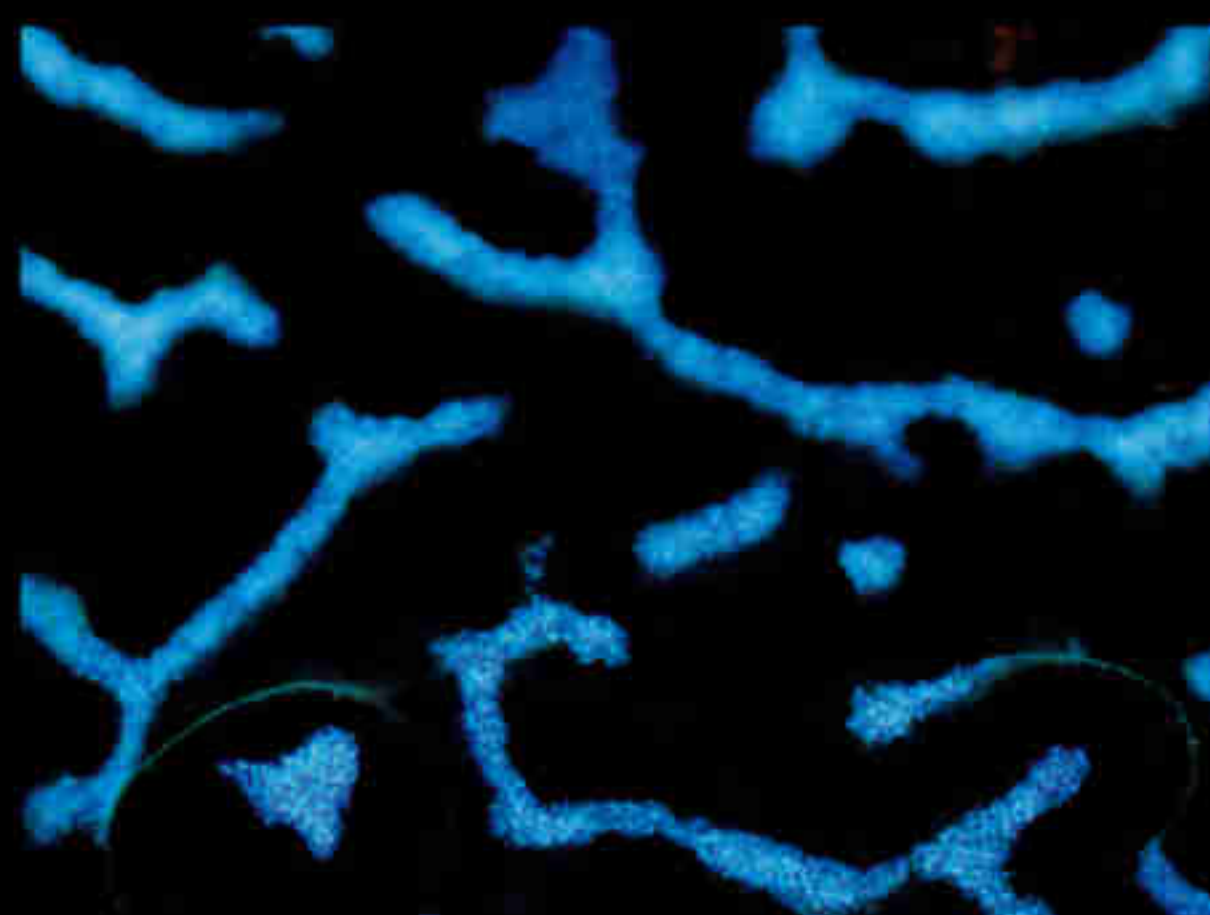


PYCNOCLAVELLA DIMINUTA

CLAM MANTLES



TRIDACNA SP.



TRIDACNA MAXIMA



TRIDACNA SIPHON



TRIDACNA SP.



TRIDACNA SP.

FLATWORMS & NUDIBRANS



NUDIBRANCH: CHROMODORIS JILLOCKI



FLATWORM: PSEUDOCEROS S



FLATWORM: PSEUDOCEROS S



NUDIBRANCH: NEMBROTHA S



FLATWORM: PSEUDOBICEROS FORDI

CRUSTACEANS



SHRIMP: RHYNCOCINETES DURBANENSIS



SHRIMP: PERICLIMENES COLEMANI



MAJID CRAB: XENOCARCINUS DEPRESSUS



CRAB: ALIAPORCELLANA SP.



REEF LOBSTER: ENOPLOMETOPUS SP.

FISH



CHAETODON MYERSI



FORCIPIGER LONGIROSTRIS



POMACANTHUS IMPERATOR



POMACANTHUS XANTHOMETOPON



NASO LITURATUS



by LES KAUFMAN
photographs by TIM LAMAN

Gaze at the vivid yellows, blues, and psychedelic swirls of a single emperor angelfish and you'll sense the whimsy of evolution.

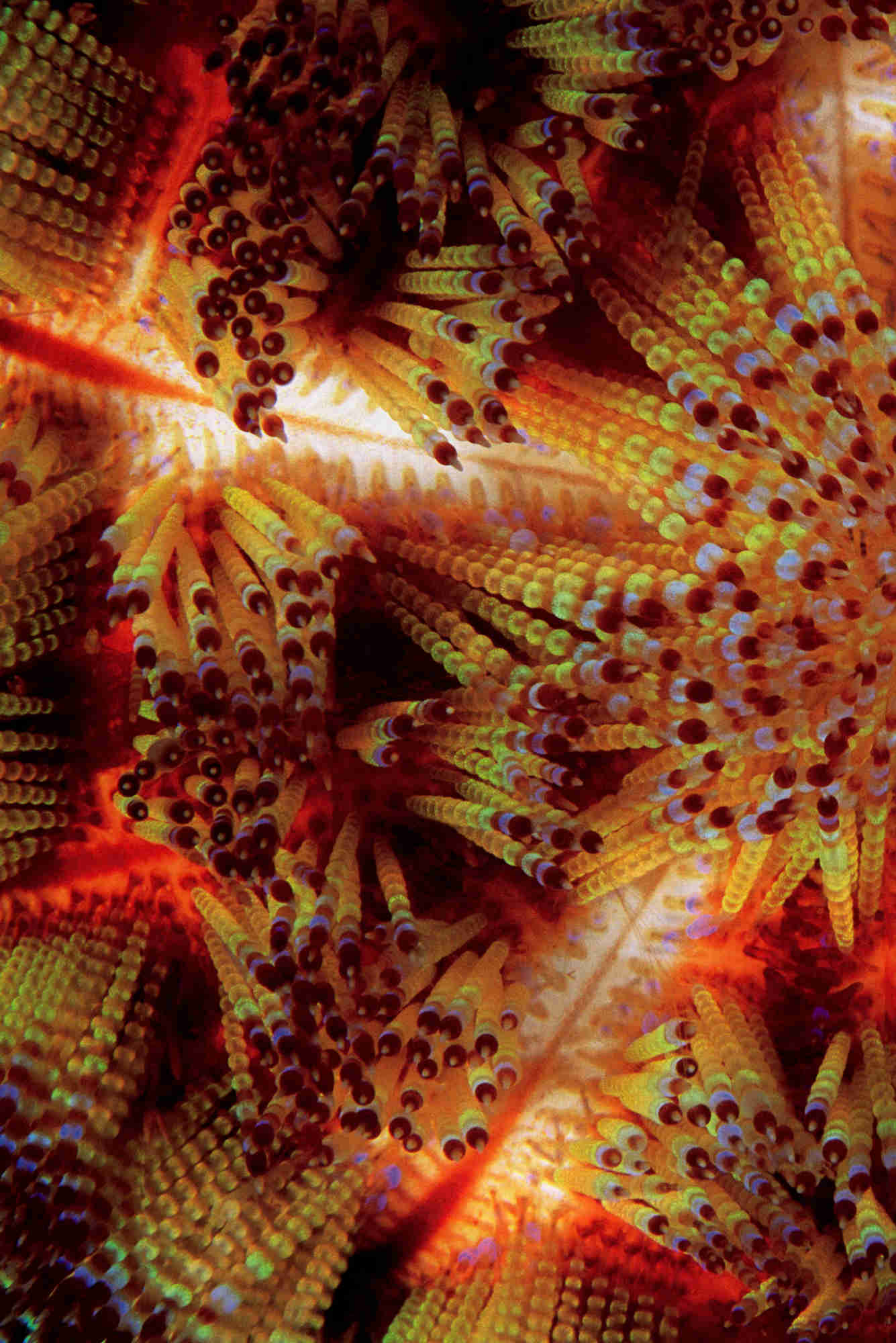
Go on to explore its home in lush coral reefs and you'll soon hit sensory overload, assaulted by colors and patterns that range from sublime to garish. Coral reefs are unquestionably the world's most colorful places. But why?


A tiny juvenile emperor angelfish swims next to an adult, showing how color and pattern change with age. As juveniles, all Indo-Pacific species of this genus, *Pomacanthus*, have similar blue-and-white swirls. Yet as they mature, each species develops its own look—a tool for attracting mates and defining territories. In the glare of a strobe, reef creatures from fish and sea stars to tunicates and worms (preceding pages) gleam with incredible hues. Now science is discovering how fish see such displays—and what messages they send.

Scientists have long known that color plays a role in sexual selection and warning of danger. But only in the past decade or so have we begun to understand how wavelengths of light (and therefore color) appear at different depths and how various marine creatures' eyes perceive this light and see each other—far differently than humans see them.

To document how reef animals use color, I joined photographer Tim Laman for a total-immersion course off Fiji and Indonesia. It was an eye-opener, with virtuoso displays of color at every turn. Beyond the world's reefs, where waters are turbid or murky, most creatures use nonvisual means of communication such as smell, taste, touch, and sound. But in the clear, sunlit waters of coral reefs, light abounds, vision predominates, and animals—both sighted and blind—drape themselves in blazing color not only to entice mates or threaten foes but also to advertise their services, evade predators, catch prey, even hide in plain sight.

Tim and I began our work by studying the healthy reef

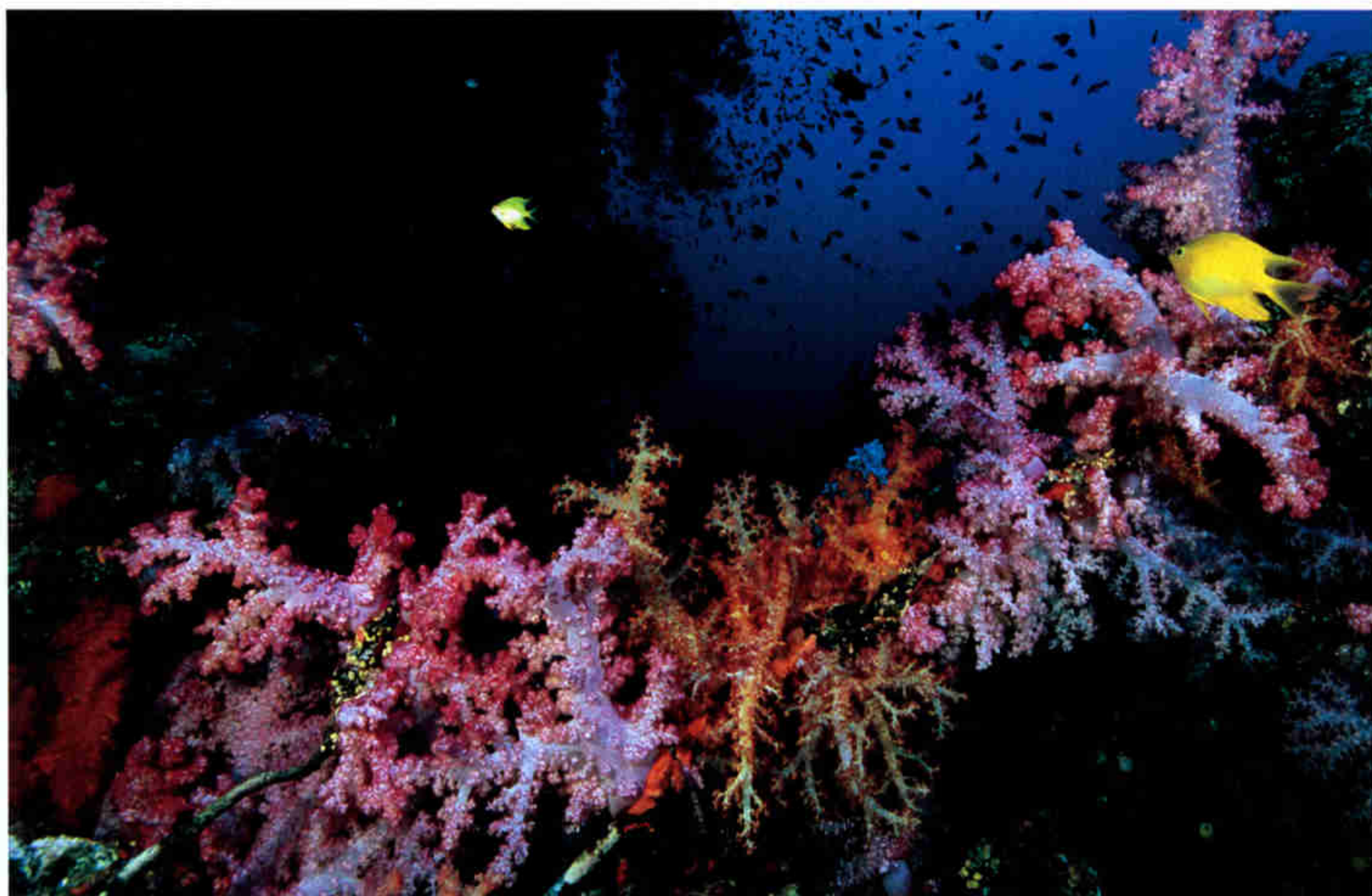




Subtle as a Vegas billboard, a fire urchin stands out at any depth, using both color and contrast to blast its message to potential predators: Steer clear, I'm toxic. Spine pricks cause a searing burn because red and bluish balls at the tips are full of venom. Such defenses allow this soft-bodied urchin to move about freely, munching algae.

ASTHENOSOMA VARIUM

When color serves as dire warning

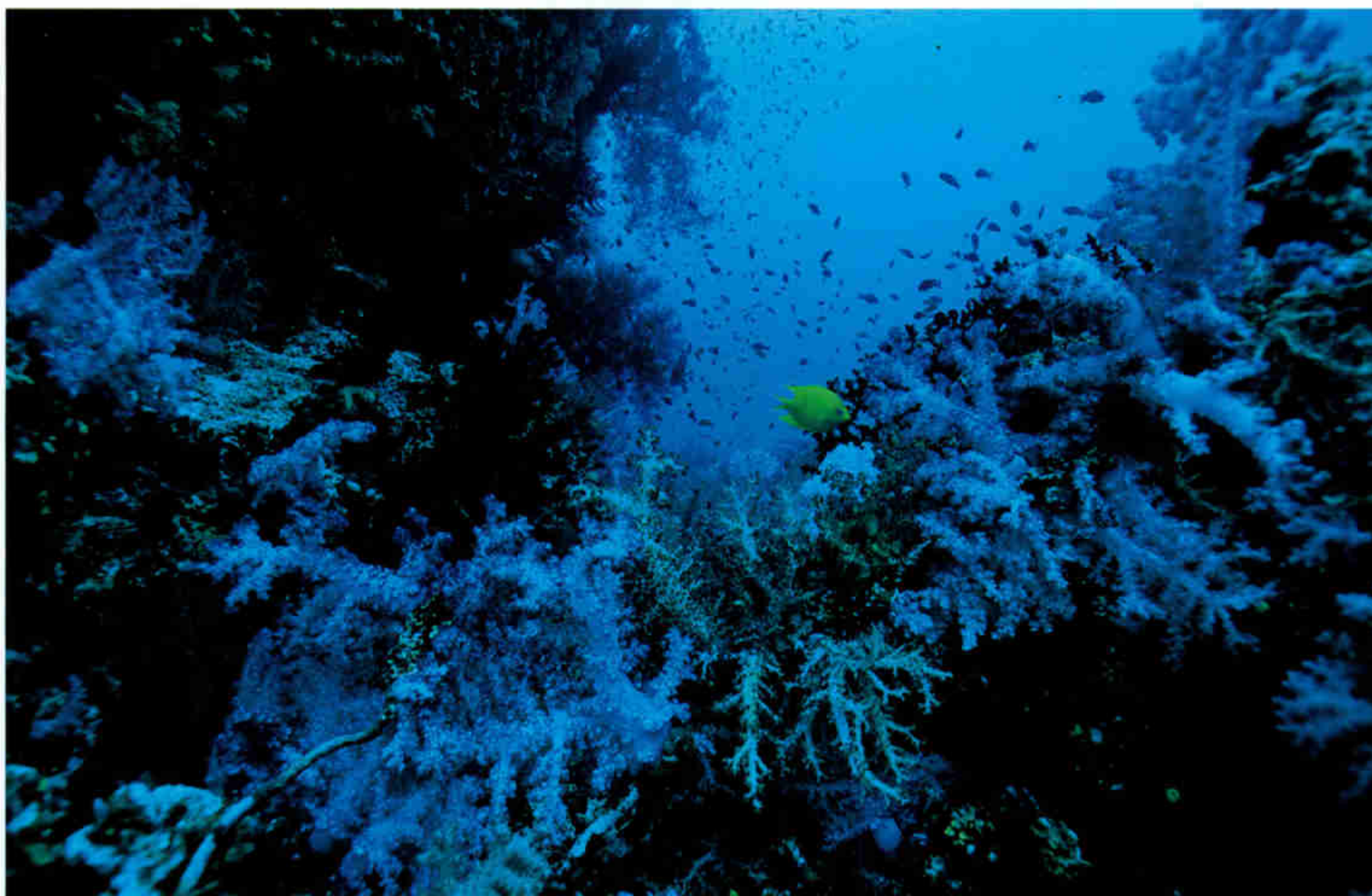


Underwater, what we see and what

systems of Fiji (NATIONAL GEOGRAPHIC, November 2004). Drifting 80 feet below the sea's surface, Tim aimed his strobe lights at a patch of reef to reveal brilliant shades of red on coral branches. But when we flicked off the artificial light, we saw the reef more as fish would see it—and it was a different world. Pale blues, greens, violets, and yellows met our gaze. The red was no longer visible, its longer wavelengths absorbed by water molecules and debris. Red pigments on marine animals may simply function as gray or black at depth; why they even have a red pigment we don't know. But we are beginning to understand more about the yellows and blues that so dominate the wardrobe of reef fish—and help make them prized targets of collectors.

Justin Marshall of the University of Queensland in Australia, George Losey of the University of Hawaii, and their colleagues study fish eyes. Using a technique called microspectrophotometry, they've analyzed the visual pigments and photosensitivity of various reef-fish eyes to determine how and what fish see. They've also measured the wavelengths of light reflected off reef features to calculate an "average reef color." It turns out that in natural light the yellows and blues that adorn many damselfish, wrasses, and angelfish blend well with that average reef background, providing camouflage from predators.

We witnessed the phenomenon of brightly colored fish hiding in plain sight throughout Indonesia, home to the highest marine diversity on Earth. In a tiny spot just southeast of Sulawesi, clouds of colored fish swam against a collage of vivid invertebrates encrusting the reef. With such an excess of pattern and color, no one creature stood out. Up close, regal angelfish flashed eye-popping bands of yellow, violet, and white. But recent studies show that as regals swim against the reef's visually complex background, their contrasting lines merge in a predator's brain. According to Boston University marine biologist Gil Rosenthal, as a reef



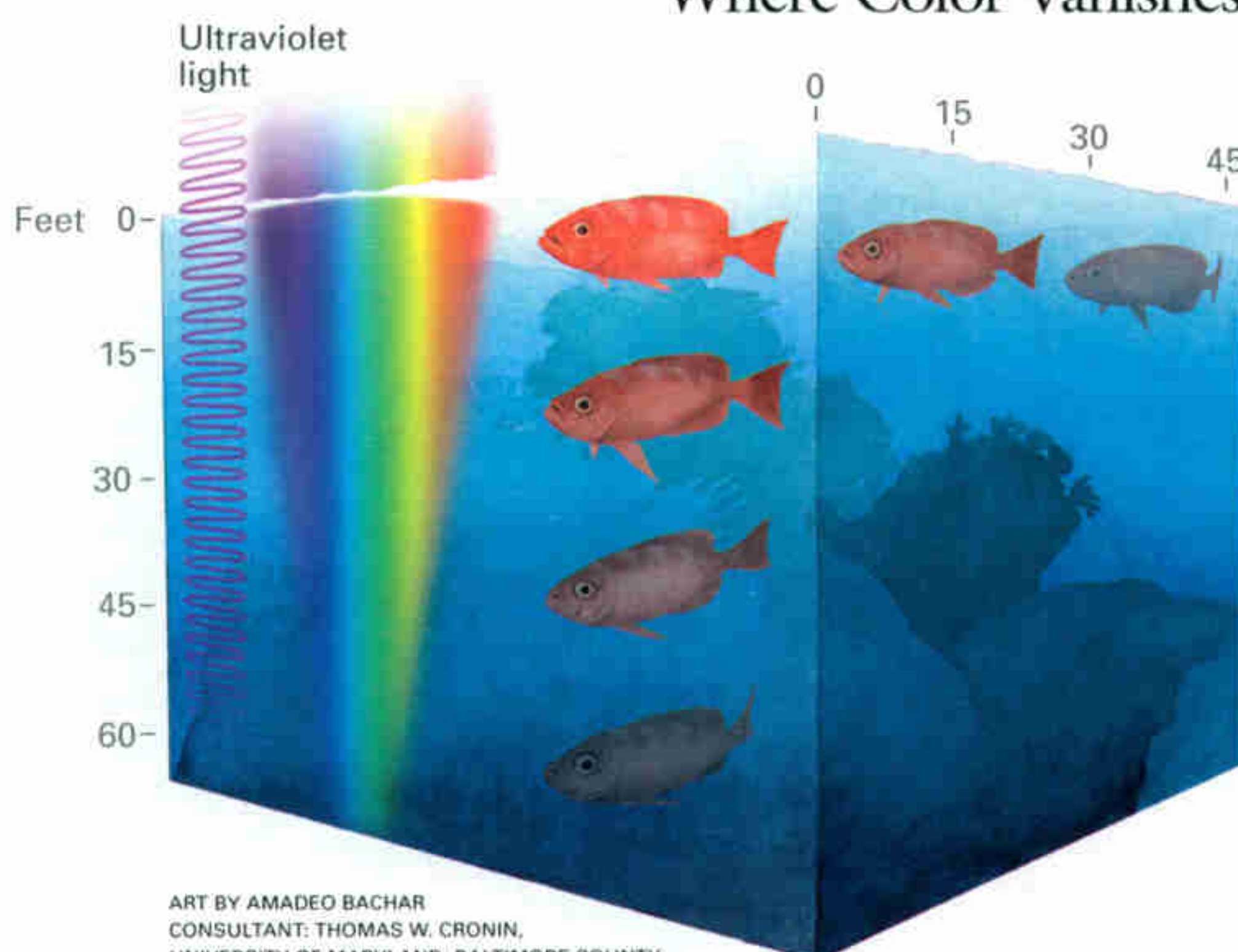
fish see can be entirely different

fish retreats, distance and motion can make it difficult for predators to perceive fine details and distinguish closely spaced outlines of contrasting colors. So at a distance, spots and stripes blur together, helping even stationary fish merge into the background of the reef and the ocean beyond.

Sulawesi is rich in cephalopods—octopuses, squids, and cuttlefish—which have the biggest brains and most mercurial colors of all the invertebrates. We got to know one octopus particularly well. It spent its days systematically moving from one outcrop to another, probing for prey with serpentine armtips thrust deep into

Behold two views of a Fijian reef, 80 feet down. In strobe light (above left) golden damselfish pop out against pink and red soft corals. But in natural light (above) red is invisible, blue dominates, and yellow blends with the reef. Absorbed by water molecules, plankton, and debris, the longer wavelengths of red dissipate at about 30 feet of vertical depth and horizontal distance (art). Shorter wavelengths of blue scatter widely—one reason why oceans look blue.

Where Color Vanishes



ART BY AMADEO BACHAR
CONSULTANT: THOMAS W. CRONIN,
UNIVERSITY OF MARYLAND, BALTIMORE COUNTY



SHRIMPS: *LYSMATA AMBOINENSIS* (ABOVE); *LYSMATA DEBELIUS*

When color says “come and get it”

Deep on a reef in Bali a *Lyсмata* shrimp plucks parasites from the mouths of waiting cardinalfish (above). Another cleans a moray eel (right). At this depth the shrimps' red bodies would look black, contrasting with their white limbs. This stark contrast—not color—may send the message: Our cleaning business is open.

coral crevices. Just before jumping to a new spot, it would darken (except for one bold white stripe), then crash to the ground with arms outstretched, the webbing between them blocking off routes of escape for small creatures such as hermit crabs caught under its body. The webbing would then turn a nearly transparent white. To us—and perhaps to trapped prey—these white patches looked like windows of light and escape. We speculate that this color-change act is a ruse to lure small, cowering animals up to the “windows” and thus toward the octopus's mouth.

When at rest, this octopus became camouflaged against the reef, with shifting patterns of dark and light on its skin that matched the texture and color of the backdrop. This appears to be an impressive trick, given that octopuses are color-blind: Their retinas lack the cells that receive and process color. But apparently these animals get by without color vision, simply responding to contrasts of shade and light.

Useful in deception, color can also speak the language of love for reef creatures. But it's a quick chat. Many reef fish can blink their colors on and off in seconds, as we saw near the coast of Bali. Rising toward the shallows through a cloud of flasher wrasses, we watched the males shoot neon blue stripes across their bodies and outstretched fins, creating a miniature laser-light show. Spurred to passion by a male's display of lights, a female rose in the water column with her chosen suitor and released an explosive burst of eggs to mix with his sperm. Job done, the male instantly went drab, and the consummated pair sped to the safety of the reef. That moment of electric bliss must have exposed them to great risk from predators,





CHEILINUS DIAGRAMMUS

Quick as a blink, coral reef fish can

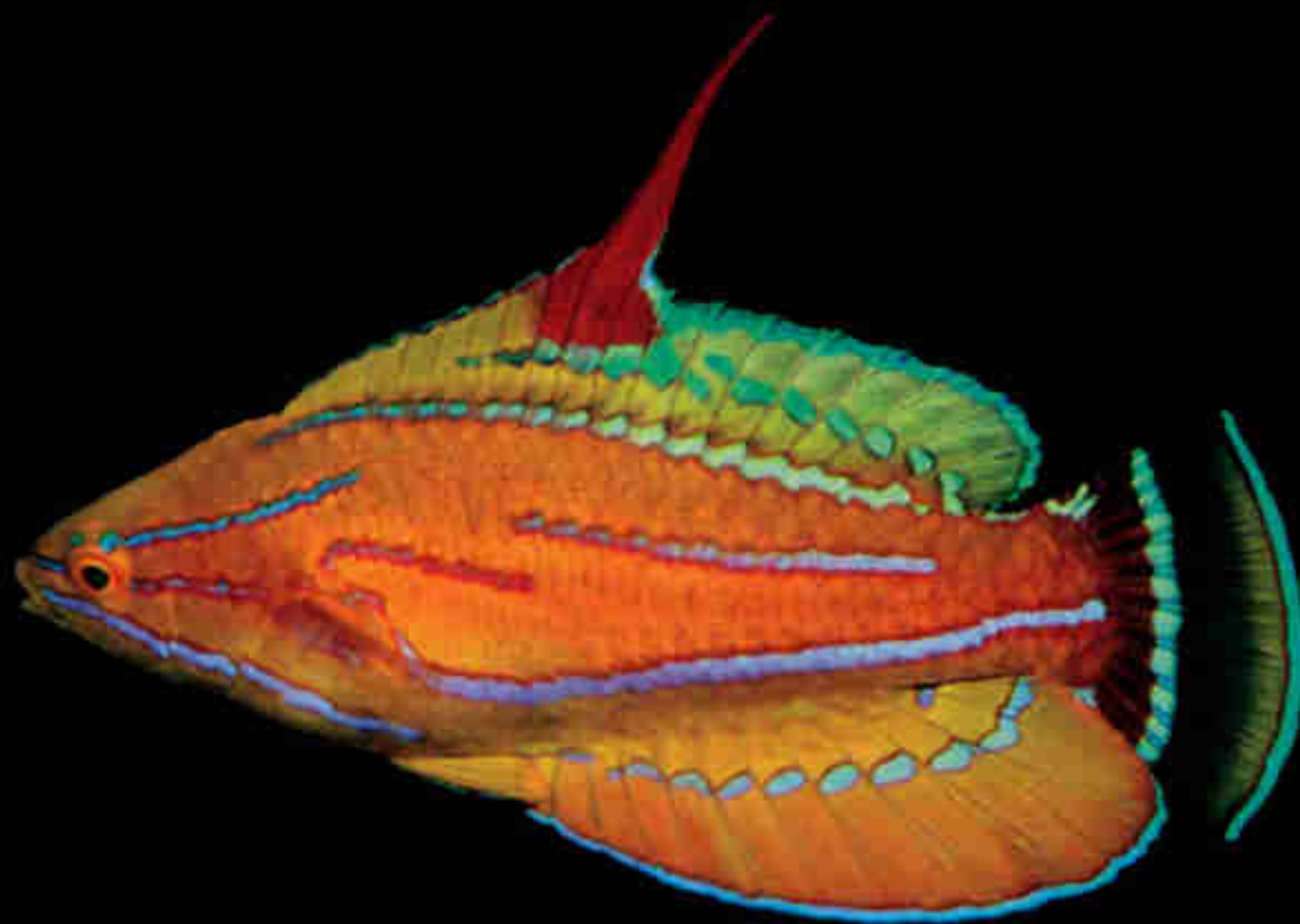
Patrolling his territory in Fiji, a barcheek wrasse (above) gapes and instantly changes color, its puffed cheeks going white, setting off black stripes. One study suggests that such yawns often precede sudden action. In this case the fish may be threatening a potential rival or warning off intruders.

so the ability to turn off color was just as important as turning it on.

The mechanism for this quick-change act is a class of skin cells called chromatophores. Controlled by both neurons and hormones, chromatophores create the appearance of color or pattern through pigments and light manipulation. Specialized chromatophores called leucophores render skin pale. To produce blue and iridescent colors like those used by the flasher wrasse, iridophores manipulate crystals of guanine, a common metabolic waste product, to scatter white light and then reflect specific wavelengths as needed. Such cells can instantly brand their bearers as terrifying, invisible, or irresistible.

With the right lighting and a bit of luck, humans can witness these vivid displays. But there's a lot that we'll never see, due to the limitations of human sight. Sailing along an island chain called Nusa Tenggara, Tim and I observed turbulence along the seam between the Pacific and Indian Oceans. This fertile mixing zone is rich with plankton, and the roiling water was jammed with plankton-feeding fish massing below the surface. We dived among great crowds of them. Clearly they were eating something—we could see their high-speed jaws flashing—but how did they spot their prey, zooplankton, which was white and all but transparent to us? Thanks to years of work by biologists George Losey, Justin Marshall, Bill McFarland, and their students, we now know that many plankton-eating fish can see ultraviolet light, which makes the zooplankton appear black and therefore more visible in the water. Humans can't see UV, and until fairly recently we thought UV light was virtually absent below the waves. We now know that UV can penetrate to depths beyond 300 feet, and that some fish not only see UV but also paint their bodies with UV reflectors to beam out messages to their kin. Damselfish, for instance, shout out to each other in UV, but their predators can't see it. Such findings make me wonder how much of the undersea world our own eyes miss.

Among the reefs' many marvels, stomatopods, or mantis shrimps, are the unrivaled visual masters, with the world's most complex eyes. Research by Marshall and marine biologists Tom Cronin, Roy Caldwell, and others has shown that stomatopod eyes have up to 16 separate kinds of light-sensing retinal cells, including four for UV light, plus sensitivity to patterns of polarization and exceptional spatial perception. (Humans have a paltry four retinal cell types and cannot see either



PARACHEILINUS FLAVIANALIS

become frightening—or beguiling

A male flasher wrasse goes from sedate to sexy (above), its flaring fins adorned with electric-blue jewels and a brazen red mast. Skin cells called iridophores reflect blue light, flashing it on and off to catch the eyes of more modestly attired females. If predators take notice, a wrasse can quickly douse the lights.

UV or polarized light.) This intricate retina delivers visual information already processed to a shrimp's tiny brain, vastly reducing the work the brain has to do to interpret its world. Those compound eyes help the smashing peacock mantis shrimp locate prey. We watched one stare intently at a spot on the reef, using its powerful arms to smash at the rock again and again to reach a target we couldn't see.

The reef is a world where vision and color are clearly a matter of life and death for those wise enough to heed the message. One day I was not so wise. Bold colors can advertise danger, and most marine biologists are not so foolhardy as to reach out and grab an unfamiliar, brilliantly colored animal. But on a languid dive near Komodo, in a forest of soft corals, I spotted a gaily colored clown crab sitting on something I didn't recognize. I ignored the something and reached for the crab, who surprised me by holding his ground, unafraid. Now I know why. He could afford to stick out like a beacon because the something he was sitting on was his form of defense—a stinging hell's fire anemone. It took two weeks for the burn marks and pain to fade from my hand. Lesson learned.

Everywhere we went in the islands, anemones and corals bore bright pastel pigments that fluoresced brilliantly orange, red, or green. The molecules that create this fluorescence could serve as sunscreens, or as light absorbers to boost growth. But in some cases these colors can be co-opted by unrelated creatures. We saw one common coral with fluorescent pink splotches, which appear on damaged spots that are healing. Fish are attracted to the pink spots and bite at them. A small parasite has evolved to infest this coral, causing harm, which leads to more pink patches that attract fish. The fish nibble the spots, thus taking up the parasite and becoming its host. Even a small parasite has developed a way to use color for its own survival.

The world's coral reefs teach that color conveys information and can change over seconds or lifetimes. It can hide or reveal, warn or beckon, broadcast widely or target a select few. Science is beginning to crack these codes—vital knowledge that will help protect reef creatures and the fragile habitats they adorn so beautifully.

EXCLUSIVE FISH'S-EYE VIEW Join photographer Tim Laman in Sights & Sounds as he explores the rainbow world of coral reefs. You can also download wallpaper and a postcard at nationalgeographic.com/magazine/0505.



A Shifting Skin

OCTOPUS SKIN CROSS SECTION
(350 MICRONS ACROSS)

Retracted
chromatophore

Leucophore

Expanded
chromatophore

ART BY AMADEO BACHAR
CONSULTANT: ROGER T. HANLON,
MARINE BIOLOGICAL LABORATORY,
WOODS HOLE, MASSACHUSETTS

On the hunt:

Drama unfolds on an Indonesian reef as an octopus goes tent-hunting: Slamming itself down on a patch of seafloor, it spreads its arms and turns



OCTOPUS CYANEA

the color of deception

the webbing a translucent bluish white. Trapped prey may dash toward these seemingly open windows of light. The octopus then gulps

them down, flicking at nearby competitors that hope to eat escapees. Skin cells called chromatophores (art, left), when triggered by

nerves and by hormones, expand and cause color change. As they retract, they expose leucophores, which make skin appear white.



Secret messages flash and vanish

In visible light a captive regal angelfish has colored bands, while in UV light the fish is black and gleams with white stripes and patterned fins. Scientists wonder why the species evolved to shimmer in UV, since it can't even see UV light.





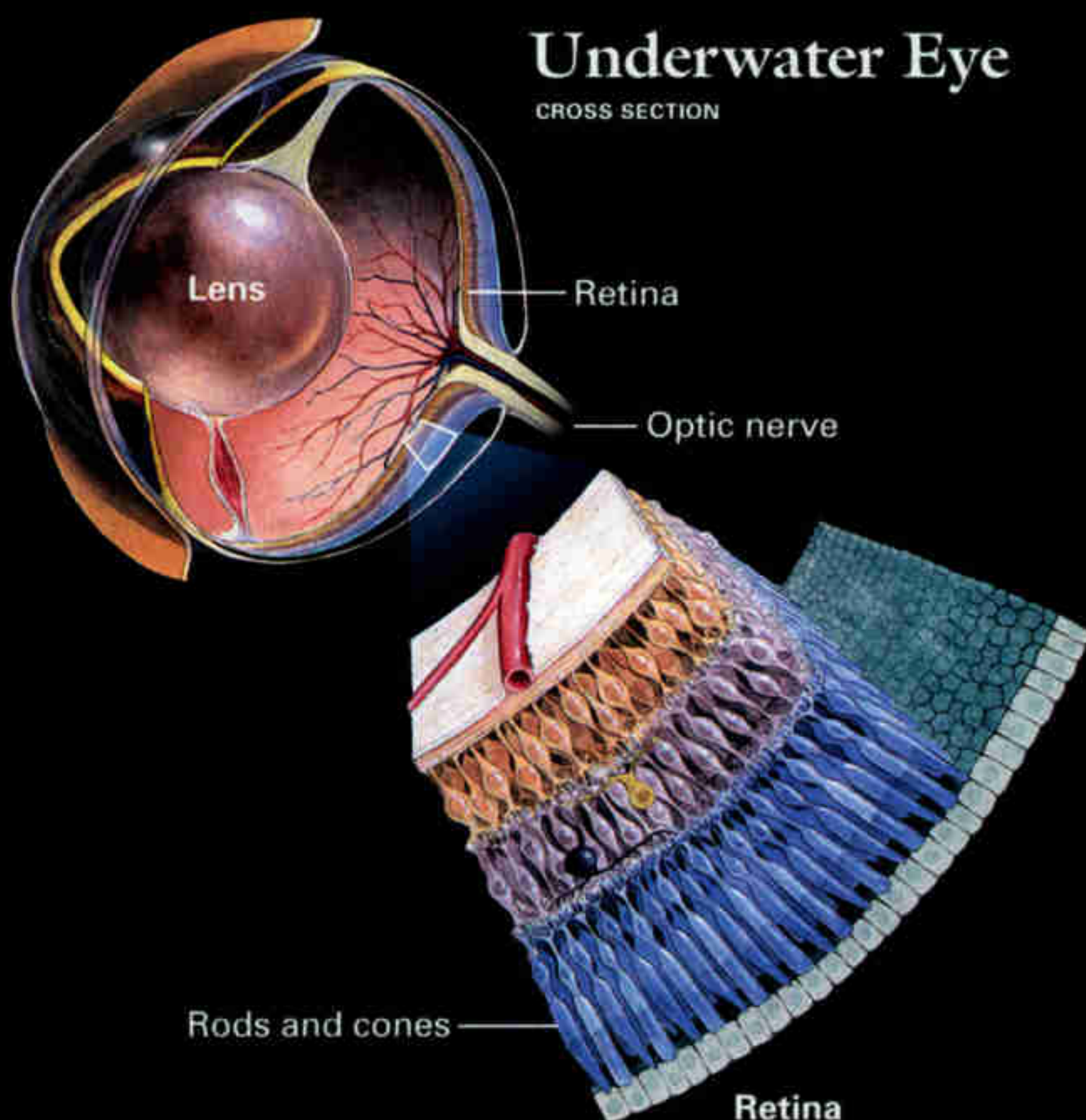
depending on the available light

Azure damselfish can see blue and yellow visible light but can also see in the UV range, where blue looks white and yellow goes black. This pattern may signal messages to friends but would elude predators, which typically can't see UV.



The pastel palette of a male squarespot anthias (left) reveals all its glory in the light of an underwater strobe. In natural light at 80 feet deep (above) this fish's violet side patch signals his virility to females and warns males to stay away. Cells in the patch make it glow in UV (above right), but it's not known yet whether this species can see UV light.

Using microspectrophotometry, scientists analyze the colors fish can detect. A fish's eye (art) is specialized to handle fickle light conditions underwater. Unlike eyes in most vertebrates, fish eyes have a totally spherical lens that corrects for refraction, or bending of light waves, in water. Cones provide sharp color vision in brighter light, while closely packed rods gather and reflect light to sharpen vision in dim conditions.

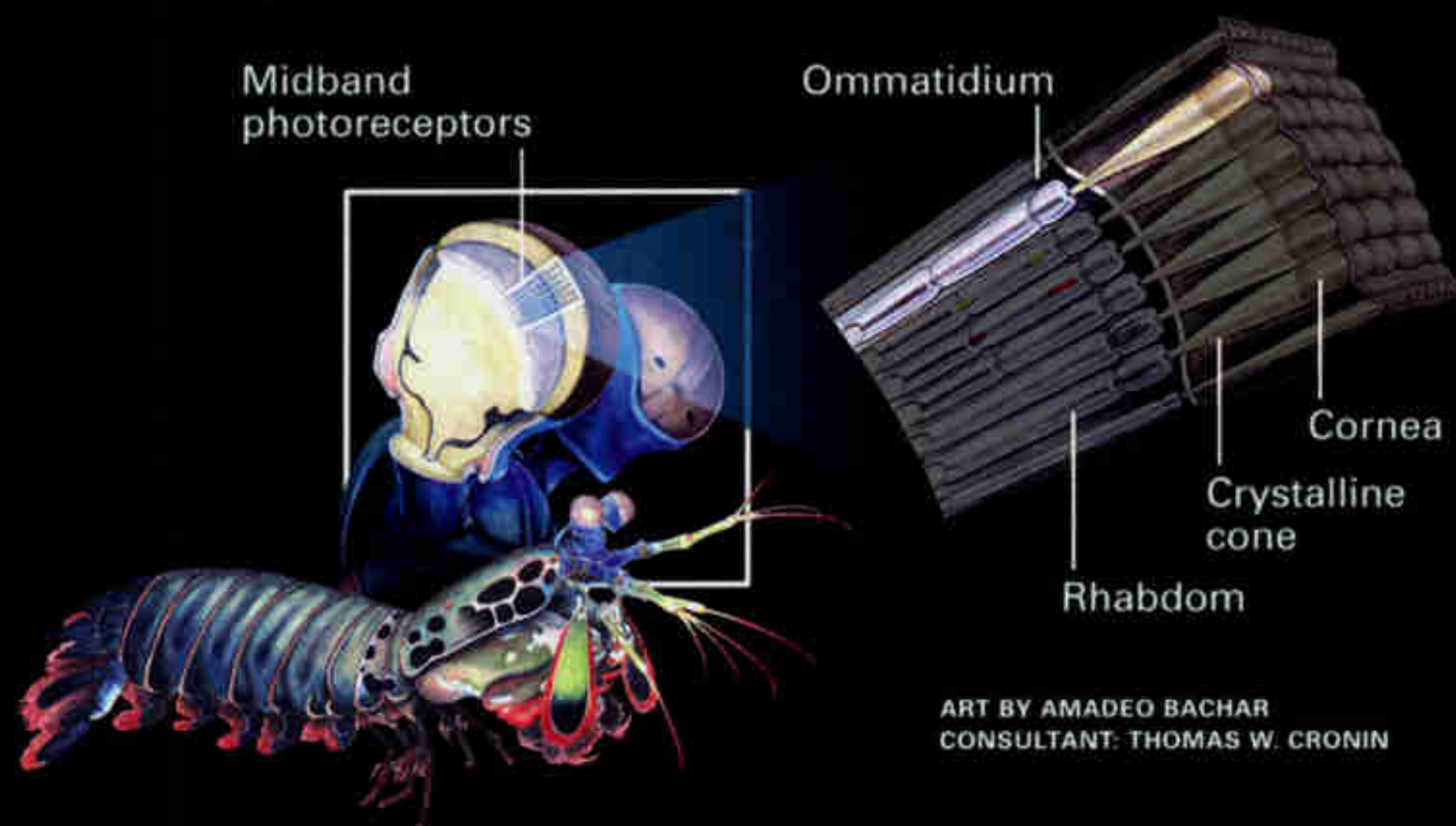


Nature's most complex eyes enable the smashing peacock mantis shrimp to detect 16 channels of light, including UV and polarized. (Humans detect only four channels.) Each tiny dome on the surface is a separate cornea that admits light. The cornea connects to a crystalline cone and a photoreceptor called a rhabdom; all three structures form what is called an ommatidium (art). The most specialized light processing occurs in the midband photoreceptors. These elaborate eyes maximize information gleaned from available light.

ODONTODACTYLUS SCYLLARUS

Mantis Microprocessor

CROSS SECTION



ART BY AMADEO BACHAR
CONSULTANT: THOMAS W. CRONIN

In the world



of eyes, these are true standouts

Bright fins,

The black sands of Indonesia grant secret refuge to a spiny devilfish (left). It spends most of its time nearly invisible against its background, lying in wait to ambush passing prey. If startled or frightened, it quickly bares the brightly decorated inner surfaces of



INIMICUS DIDACTYLUS (ABOVE AND RIGHT)



DENDROCHIRUS ZEBRA



DENDROCHIRUS BIOCELLATUS



SIGNIGOBIOUS BIOCELLATUS



fake eyes, and stealth aid survival

its pectoral fins (below), warning intruders of its venomous spines. It will also flash this display in hopes of persuading a potential mate of its vigor.

Closely related, the zebra lionfish and twinspot lionfish (two center photos at left)

also exhibit species-specific pectoral fins when threatened or amorous. Lionfish also seem to use these flags to confuse and herd prey.

Like the twinspot lionfish, crabeye gobies (bottom left) have fin markings called double eyespots that mimic huge

eyes. These 'eyes' closely match the size and spacing of eyes on fish like groupers, which eat the fish that eat gobies. So this is a tricky way for tiny gobies to scare off potential predators—by making them think they're facing their own worst enemies.



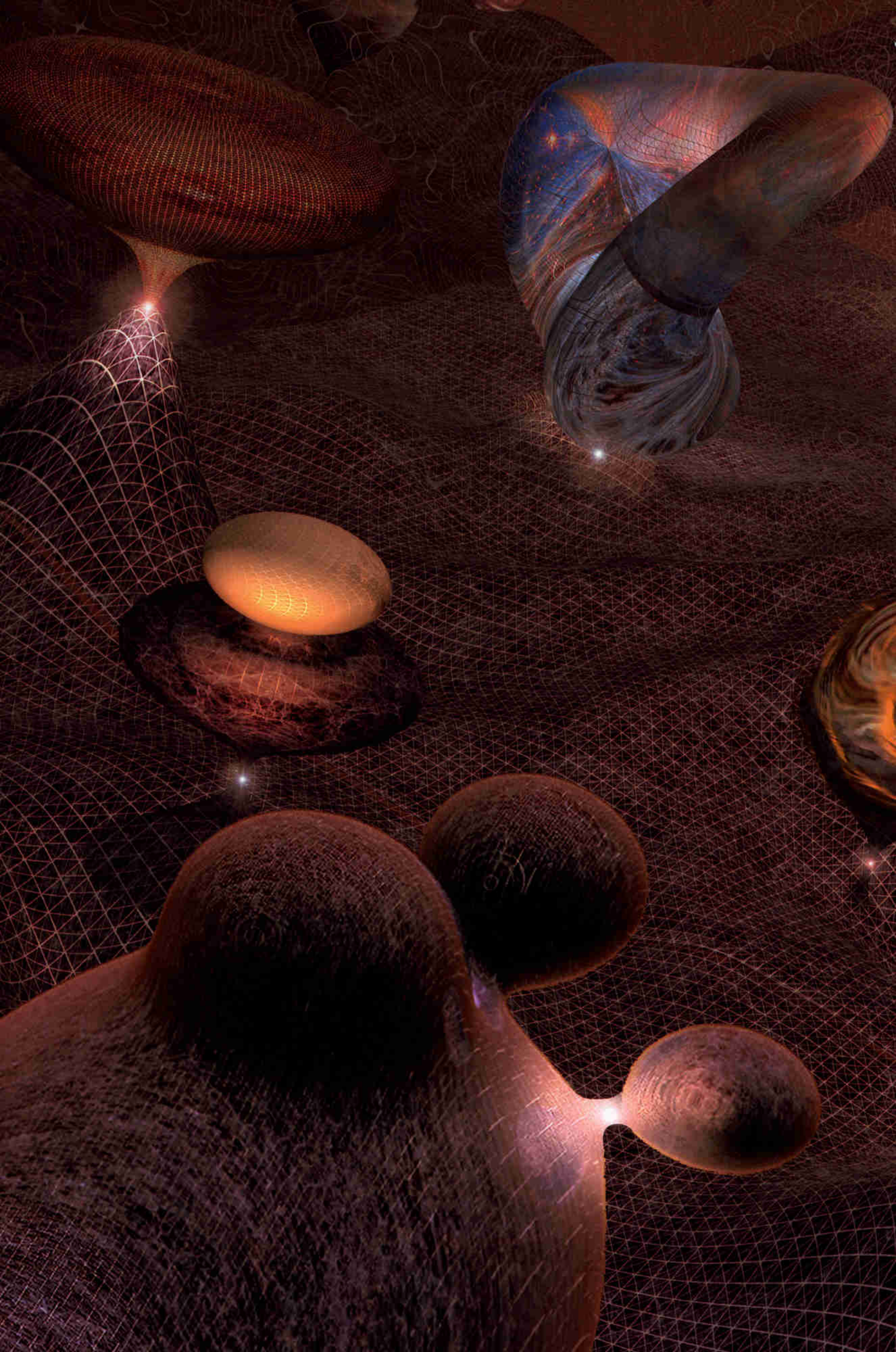
Where sex change is nature's way

Deep maroon marks a spine-cheek anemonefish as the dominant female of this host anemone, where she rests with her orange mate. When she dies, he will turn maroon, become female, and mate with the next male in line. This color-coded sex change vividly shows that in the drama of coral reefs, color plays a leading role. □

PREMNAS BIACULEATUS









origin

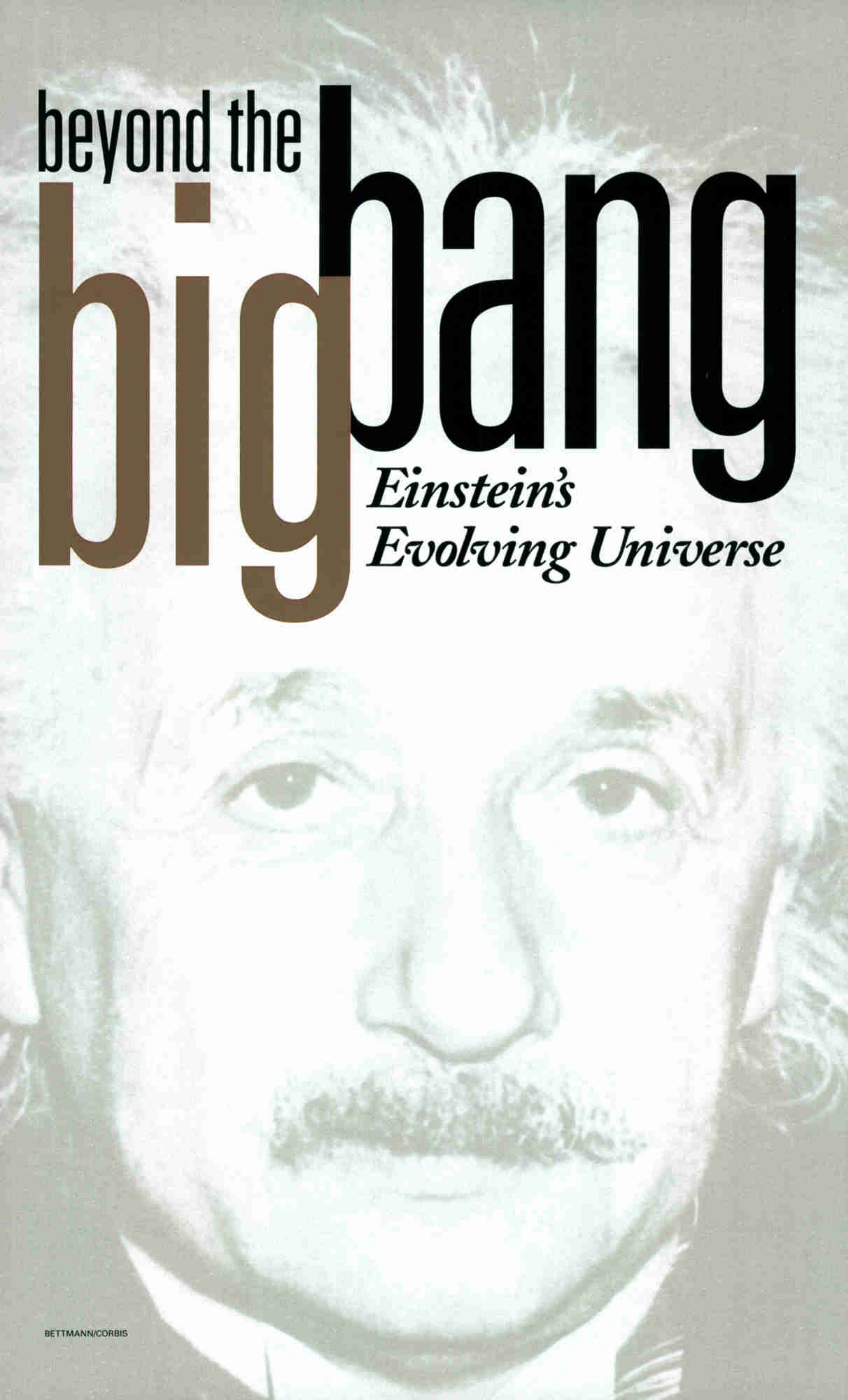
Infinite Beginnings

Pushing the limits of theory and imagination in true Einsteinian

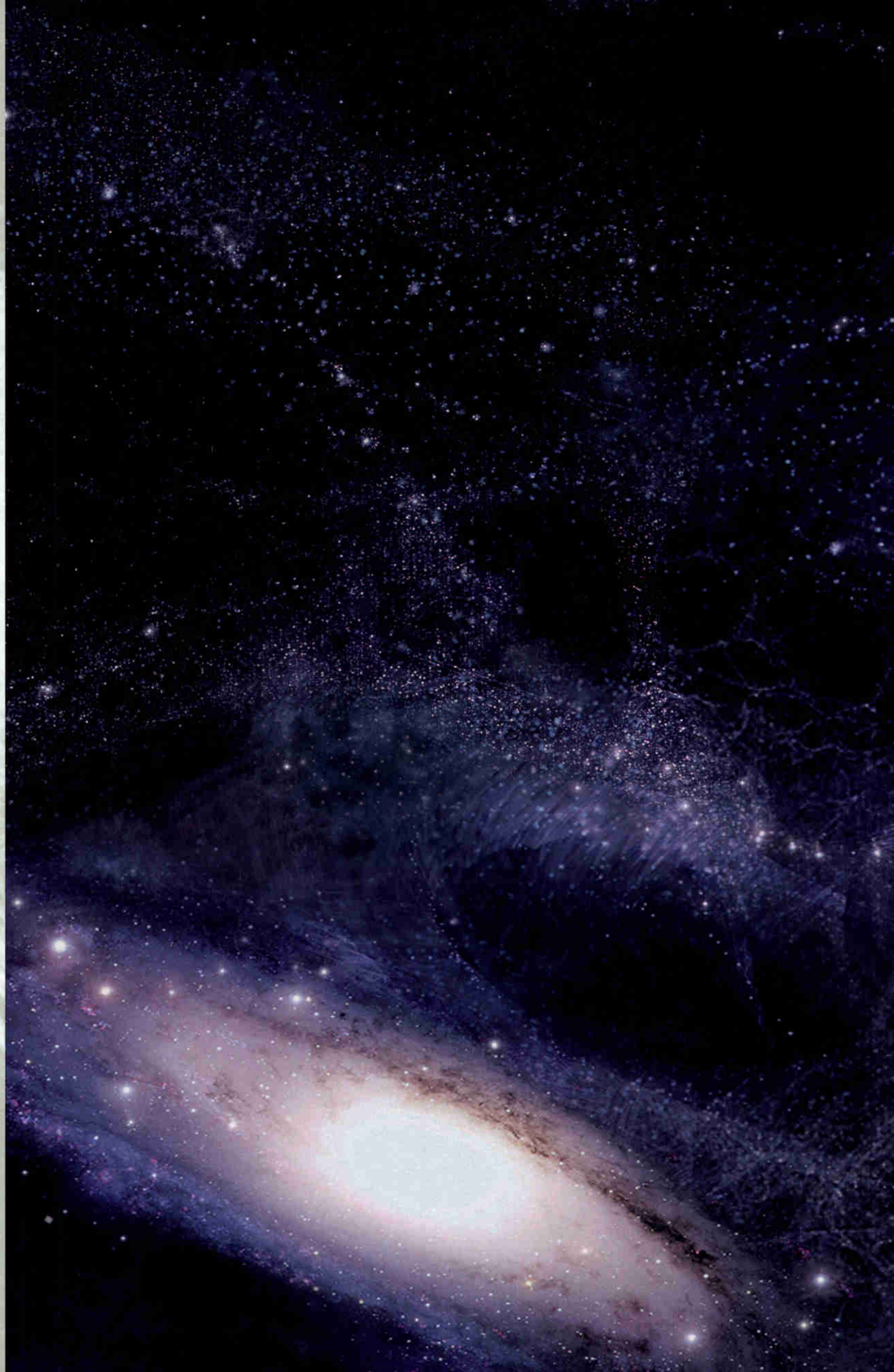
fashion, cosmologists are daring to speculate that ours is not the only universe. The big bang that created everything we know of space and time could be just one of an infinite number of beginnings, yielding a never ending sequence of universes.

The scenario, shown in this artist's concept, emerges from inflation theory, a descendant of Einstein's general theory of relativity. Relativity implies that space and time can stretch to vast dimensions from a tiny starting point; inflation describes how our own universe ballooned in its first moments and suggests that the same thing can happen anywhere, at any time. The result: an eternal expanse

of space erupting with bubbles of energy, or big bangs, each the seed of a universe. Not all universes will be alike. While a cosmos like our own glows with galaxies (at lower right), others may contain more dimensions or different forms of matter. In some, even the laws of physics work differently (twisted universe at upper left).



beyond the
big bang
*Einstein's
Evolving Universe*





structure

The Invisible Web

Something out there holds swarms of galaxies together and keeps their stars

from flying apart, but scientists still haven't learned what this invisible substance is. Known as dark matter, it gathers to form a colossal cosmic scaffolding. Astronomers believe that galaxies formed at the densest points in this weblike structure, and the dark matter continues to hold them in place with its gravity. Its bulky presence can be detected by tracking stars on the outskirts of galaxies, which move at speeds that would be impossible if only visible matter—a galaxy's other stars and gas—were pulling on them. Astronomers have also mapped this unseen substance with the help of an effect predicted by Einstein's general relativity: Dark matter's gravity wrinkles space-time, bending light rays as they pass. Such measurements indicate that dark matter could make up 90 percent of the universe's total mass.

These days, cosmologists are searching for the identity of dark matter, trying to detect the elusive substance responsible for arranging everything we see in the sky.

By Marcia Bartusiak
Art by Moonrunner Design

On January 29, 1931, the world's premier physicist, Albert Einstein, and its foremost astronomer, Edwin Hubble, settled into the plush leather seats of a sleek Pierce-Arrow touring car for a visit to Mount Wilson in southern California. They were chauffeured up the long, zigzagging dirt road to the observatory complex on the summit, nearly a mile above Pasadena. Home to the largest telescope of its day, Mount Wilson was the site of Hubble's astronomical triumphs. In 1924 he had used the telescope's then colossal 100-inch mirror to confirm that our galaxy is just one of countless "island universes" inhabiting the vastness of space. Five years later, after tracking the movements of these spiraling disks, Hubble and his assistant, Milton Humason, had revealed something even more astounding: The universe is swiftly expanding, carrying the galaxies outward.

On the peak that bright day in January, the 51-year-old Einstein delighted in the telescope's instruments. Like a child at play, he scrambled about the framework, to the consternation of his hosts. Nearby was Einstein's wife, Elsa. Told that the giant reflector was used to determine the universe's shape, she reportedly replied, "Well, my husband does that on the back of an old envelope."

That wasn't just wifely pride. Years before Hubble detected cosmic expansion, Einstein had fashioned a theory, general relativity, that could explain it. In studies of the cosmos, it all goes back to Einstein.

Just about anywhere astronomers' observations take them—from the nearby sun to the black holes in distant galaxies—they enter Einstein's realm, where time is relative, mass and energy are interchangeable, and space can stretch and warp. His footprints are deepest in cosmology, the study of the universe's history and fate. General relativity "describes how our universe was born, how it expands, and what its future will be," says Alan Dressler of the Carnegie Observatories. Beginning, middle, and end—"all are connected to this grand idea."

At the turn of the 20th century, 30 years before Einstein and Hubble's rendezvous at Mount Wilson, physics was in turmoil. X-rays, electrons, and radioactivity were just being discovered, and physicists were realizing that their trusted laws of motion, dating back more than 200 years to Isaac Newton, could not explain how these strange new particles flit through space. It took a rebel, a cocky kid who spurned rote learning and had an unshakable faith in his own abilities, to blaze a trail through this baffling new territory. This was not the iconic Einstein—the sockless, rumpled character with baggy sweater and fright-wig coiffure—but a younger, more romantic figure with alluring brown eyes and wavy hair. He was at the height of his prowess.

Among his gifts was a powerful physical instinct, almost a sixth sense for knowing how nature should work. Einstein thought in images, such as one that began haunting him as a teenager: If a man could keep pace with a beam of light, what would he see? Would he see the electromagnetic wave frozen in place like some glacial swell? "It does not seem that something like that can exist!" Einstein later recalled thinking.

He came to realize that since all the laws of

physics remain the same whether you're at rest or in steady motion, the speed of light has to be constant as well. No one can catch up with a light beam. But if the speed of light is identical for all observers, something else has to give: absolute time and space. Einstein concluded that the cosmos has no universal clock or common reference frame. Space and time are "relative," flowing differently for each of us depending on our motion.

Einstein's special theory of relativity, published a hundred years ago, also revealed that energy and mass are two sides of the same coin, forever linked in his famed equation $E = mc^2$. (E stands for energy, m for mass, and c for the speed of light.) "The idea is amusing and enticing," wrote Einstein, "but whether the Almighty is . . . leading me up the garden path—that I cannot know." He was too modest. The idea that mass could be transformed into pure energy later helped astronomers understand the enduring power of the sun. It also gave birth to nuclear weapons.

But Einstein was not satisfied. Special relativity was just that—special. It could not describe all types of motion, such as objects in the grip of gravity, the large-scale force that shapes the universe. Ten years later, in 1915, Einstein made up for the omission with his general theory of relativity, which amended Newton's laws by redefining gravity.

General relativity revealed that space and time are linked in a flexible four-dimensional fabric that is bent and indented by matter. In this picture, Earth orbits the sun because it is caught in the space-time hollow carved by the sun's mass, much as a rolling marble would circle around a bowling ball sitting in a trampoline. The pull of gravity is just matter sliding along the curvatures of space-time.

Einstein shot to the pinnacle of celebrity in 1919, when British astronomers actually measured this warping. Monitoring a solar eclipse, they saw streams of starlight bending around the darkened sun. "Lights All Askew in the Heavens. Stars Not Where They Seemed or Were Calculated to be, but Nobody Need Worry," proclaimed the headline in the *New York Times*.

With this new insight into gravity, physicists at last were able to make actual predictions about the universe's behavior, turning cosmology into a science. Einstein was the first to try. Yet as events showed, even Einstein was a fallible genius. A misconception about the nature of the

universe led him to propose a mysterious new gravitational effect—a notion he soon rejected. But he may have been right for the wrong reasons, and his "mistake" may yet turn out to be one of his deepest insights.

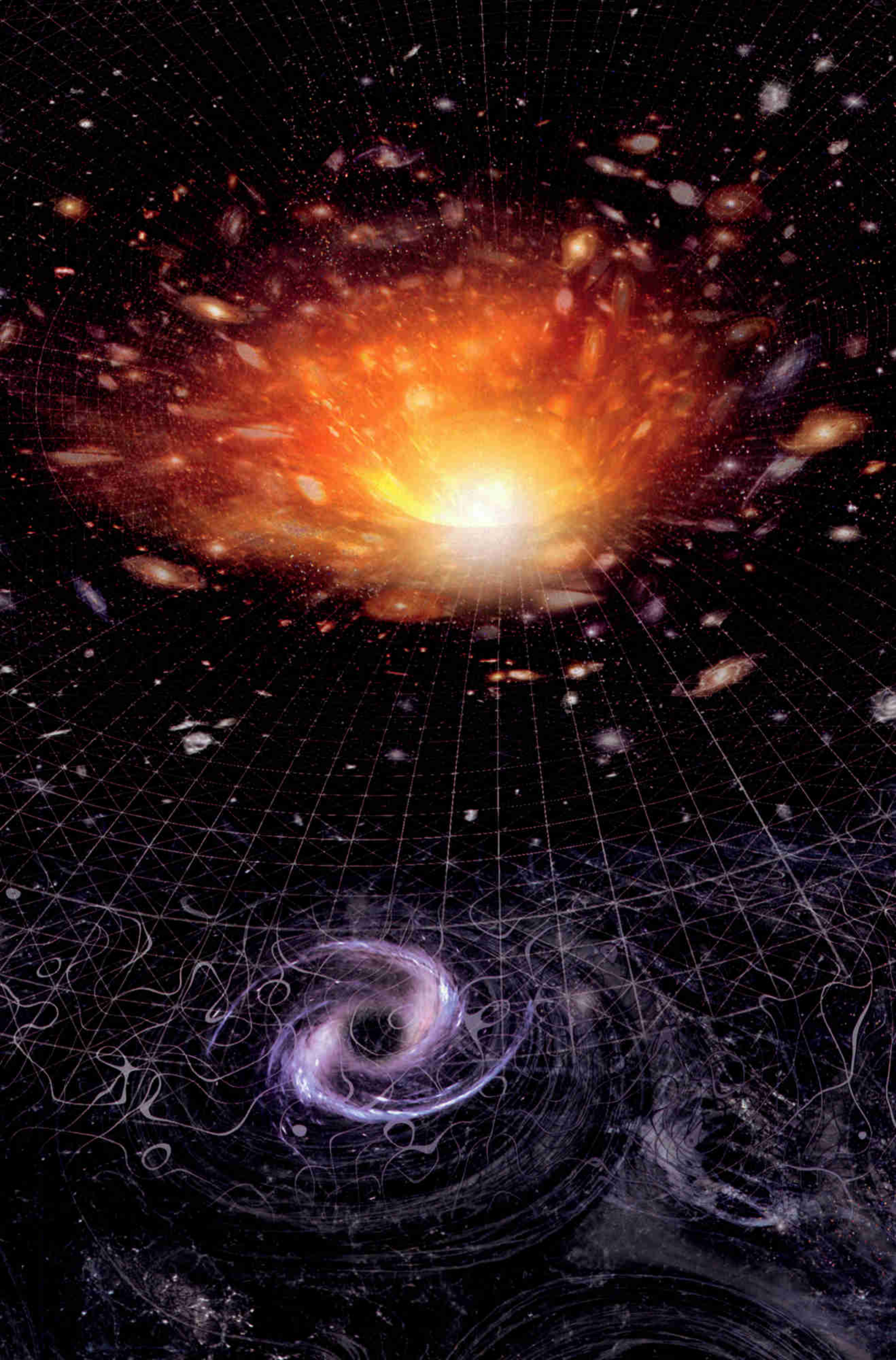
For Newton, space was eternally at rest, merely an inert stage on which objects moved. But with general relativity, the stage itself became an active player. The amount of matter within the universe sculpts its overall curvature. And space-time itself can be either expanding or contracting.

When Einstein announced general relativity in 1915, he could have taken the next step and declared that the universe was in motion, more than a decade before Hubble directly measured cosmic expansion. But at the time, astronomers conceived of the universe as a large collection of stars fixed forever in the void. Einstein accepted this immutable cosmos. Truth be told, he liked it. Einstein was often leery of the most radical consequences of his ideas.

But because even a static universe would eventually collapse under its own gravity, he had to slip a fudge factor into the equations of general relativity—a cosmological constant. While gravity pulled celestial objects inward, this extra gravitational effect—a kind of antigravity—pushed them apart. It was just what was needed to keep the universe immobile, "as required by the fact of the small velocities of the stars," Einstein wrote in 1917.

Twelve years later, Hubble's discovery of other galaxies racing away from ours, their light waves stretched and reddened by the expansion of space-time, vanquished the static universe. It also eliminated any need for a cosmological constant to hold the galaxies steady. During his 1931 California visit, Einstein acknowledged as much. "The red shift of distant nebulae has smashed my old construction like a hammer blow," he declared. He reputedly told a colleague that the cosmological constant was his biggest blunder.

With or without that extra ingredient, the basic recipe for the expanding universe was Einstein's. But it was left to others to identify one revolutionary implication: a moment of cosmic creation. In 1931 the Belgian priest and astrophysicist Georges Lemaître put the fleeing galaxies into reverse and imagined them eons ago merged in a fireball of dazzling brilliance—a



The background of the entire page is a deep space image showing a complex web of dark matter filaments and voids, known as the cosmic web. In the lower-left quadrant, there is a detailed illustration of a galaxy cluster, with several bright, glowing galaxies and a central region of intense light and energy. The overall color palette is dark, with blues, purples, and oranges from the celestial bodies.

expansion

Fast Forward: The Big Rip?

The death of the universe could rival its birth in explosive drama

if a puzzling form of energy continues to accelerate the expansion of space-time. Since the 1920s astronomers have thought the expansion was slowing down, but recent observations of distant stars reveal that the stretching of space is actually speeding up. If it picks up even more, the universe could be headed for a “big rip.” An artist’s conception of this scenario—one of many possible fates—shows how, some 20 billion years from now, unchecked expansion could tear matter apart, from galaxies all the way down to atoms. The driving force is a mysterious “dark energy” that counteracts gravity’s pull and might ultimately defeat all the forces that bind matter. Einstein was the first to introduce the notion of repulsive gravity, but he later disavowed it. Dark energy, says cosmologist Michael S. Turner, who coined the term, “has the destiny of the universe in its hands.” Although we live in the best of times, under a sky full of stars, it will grow ever darker and emptier as space-time expands.

“primeval atom,” as he put it. “The evolution of the world can be compared to a display of fireworks that has just ended: some few red wisps, ashes and smoke,” wrote Lemaître. From this poetic scenario arose today’s big bang.

Many were appalled by this concept. “The notion of a beginning . . . is repugnant to me,” said British astrophysicist Arthur Eddington in 1931. But evidence in its favor slowly gathered, climaxing in 1964, when scientists at Bell Telephone Laboratories discovered that the cosmos is awash in a sea of microwave radiation, the remnant glow of the universe’s thunderous launch. Ever since then the image of the big bang has shaped and directed the work of cosmologists as strongly as Ptolemy’s celestial spheres influenced astronomers in the Middle Ages.

Freedman and others confidently peg the universe’s current rate of expansion, as well as its age. A birthday cake for the universe would require some 14 billion candles.

Astronomers have found some strange objects in this expanding universe—and these too are Einstein’s children. In the 1930s a young Indian physicist, Subrahmanyan Chandrasekhar, applied special relativity and the new theory of quantum mechanics to a star. He warned that if it surpassed a certain mass, it would not settle down as a white dwarf at the end of its life (as our sun will). Instead gravity would squeeze it down much further, perhaps even to a singular point. Horrified, Eddington declared that “there should be a law of Nature to

Now Einstein’s “biggest blunder” is starting

In 1980 Alan Guth, now at the Massachusetts Institute of Technology, gave the big bang a boost, adding new particle physics to Einstein’s flexible space-time. He realized that for its first trillionth of a trillionth of a second, the infant cosmos could have undergone a supercharged expansion—an instant of “inflation”—before settling into more measured growth.

Inflation would have helped smooth out the matter and energy in the universe and flattened its overall space-time curvature, just as satellites have found by making precise measurements of the cosmic microwaves. And these days some theorists believe inflation wasn’t a flash in the pan. In an ongoing process of creation, space-time could be inflating into new universes everywhere and all the time—an infinity of big bangs.

Within our own universe, the high priests of astronomy have continued the cosmological quest initiated by Einstein and Hubble, first at Mount Wilson, then at the 200-inch telescope on California’s Palomar Mountain, 90 miles to the south. How fast is the universe ballooning outward? they asked. How old is it? “Answering those questions,” says Wendy Freedman, director of the Carnegie Observatories, “turned out to be more difficult than anyone anticipated.”

Only at the turn of this century, with the help of a space telescope aptly named Hubble, did

prevent a star from behaving in this absurd way!”

There was no such law. Chandrasekhar had opened the door for others to contemplate the existence of the most bizarre stars imaginable. First there was a naked sphere of neutrons just a dozen miles wide born in the throes of a supernova, the explosion of a massive star. A neutron star’s density would be equivalent to packing all the cars in the world into a thimble. Then there was the peculiar object formed from the collapse of an even bigger star or a cluster of stars—enough mass to dig a pit in space-time so deep nothing can ever climb out.

Einstein himself tried to prove that such an object—a black hole, it was later christened—could not exist. Like Eddington, he loathed what would be found at a black hole’s center: a point of zero volume and infinite density, where the laws of physics break down. The discoveries that might have forced him to acknowledge his theory’s strange offspring came after his death in 1955.

Astronomers identified the first quasar, a remote young galaxy disgorging the energy of a trillion suns from its center, in 1963. Four years later, much closer to home, observers stumbled on the first pulsar, a rapidly spinning beacon emitting staccato radio beeps. Meanwhile spaceborne sensors spotted powerful x-rays and gamma rays streaming from points around the sky.

All these new, bewildering signals are believed to pinpoint collapsed objects—neutron stars and black holes—whose crushing gravity and dizzying spin turn them into dynamos. With their discovery, the once sedate universe took on an edge; it metamorphosed into an Einsteinian cosmos, filled with sources of titanic energies that can be understood only in the light of relativity.

Even Einstein's less celebrated ideas have had remarkable staying power. As early as 1912 he realized that a faraway star can act like a giant spyglass, its gravity deflecting passing light rays and magnifying objects behind it. He eventually concluded that this tiny effect defied "the resolving power of our instruments" and had "little value."

With today's telescopes, astronomers are seeing galaxies and galaxy clusters act as powerful

effect—now called dark energy—is big enough, it could also drive the acceleration. "The need came back, and the cosmological constant was waiting," says Adam Riess of the Space Telescope Science Institute, one of the discoverers of the acceleration. "It's totally an Einsteinian concept."

So is a prediction of general relativity that, if confirmed, could open new insights into the cosmos: ripples in space-time called gravity waves. To detect them, physicists have built three giant sensors, in south-central Washington State, Louisiana, and south of Pisa, Italy. In each one, laser beams run up and down miles-long pipes to measure the slight stretching and squeezing of space-time expected if a gravity wave passes by.

By triangulating these measurements, scientists might trace gravity waves back to their sources.

to look like one of his greatest successes.

gravitational lenses, offering a peek at galaxies farther out. Since the light-bending depends on the mass of the lens, the effect also lets observers weigh the lensing galaxies. They turn out to have far more mass than can be seen. It's part of the universe's mysterious dark matter, the roughly 90 percent of its mass that can't be found in stars, gas, planets, or any other known form of matter.

A cosmic web of dark matter is now thought to have governed where galaxies formed. Dark matter is the universe's hidden architecture, and gravitational lensing is one of the few practical ways to "see" it. An effect Einstein thought insignificant has become a key astronomical tool.

Theorists have also dusted off his discarded cosmological constant to explain a startling new discovery, and now Einstein's "biggest blunder" is starting to look like one of his greatest successes. Astronomers had assumed that gravity is gradually slowing the expansion of the universe. But in the late 1990s two teams, measuring the distances to faraway exploding stars, found just the opposite. Like buoy markers spreading apart on ocean currents, these supernovae revealed that space-time is ballooning outward at an accelerating pace.

For Einstein, the cosmological constant was a way to steady the universe. But if its repulsive

Only stunningly violent events could cause space-time to shudder—a supernova, for example, or the titanic collision of two neutron stars or black holes. "If two black holes collided, gravity waves would be the only signals to come out," says Adalberto Giazotto, a scientist with the Pisa project.

The mighty jolt of cosmic birth probably also generated gravity waves, which would still be resonating through the cosmos. These remnant ripples could hold direct evidence of the fleeting moment when physicists believe all of nature's forces were united. If so, Einstein's gravity waves could at last offer clues to something he tried and failed to develop: a "theory of everything." Physicists are still seeking such a theory—a single explanation for both the large-scale force of gravity and the short-range forces inside the atom.

Catching these faint echoes of the big bang is a major goal of NASA's next generation of space astronomy missions, a plan the agency has tagged "Beyond Einstein."

Beyond Einstein? Not by a long shot. Einstein might be startled by the universe as we understand it today. But it is unmistakably his. □

HOW DOES EARTH WARP SPACE-TIME? A satellite holding the world's most perfect sphere is giving Einstein's predictions a tough test. Learn about Gravity Probe B and NASA's Beyond Einstein program at nationalgeographic.com/magazine/0505.

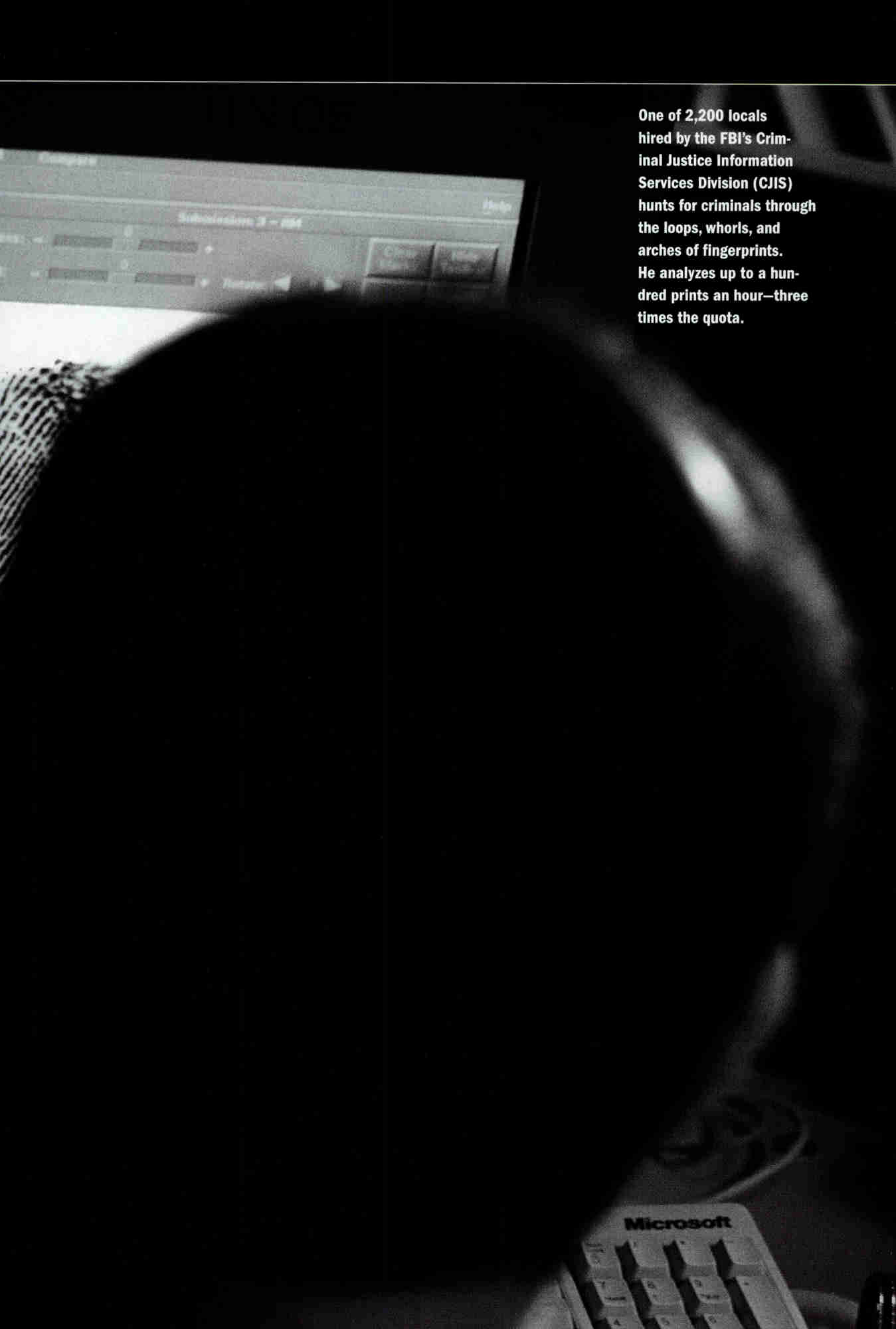


26306

WHERE THE CRIME CLOCK NEVER STOPS

FBI

BY PETER MEYER PHOTOGRAPHS BY NINA BERMAN



One of 2,200 locals hired by the FBI's Criminal Justice Information Services Division (CJIS) hunts for criminals through the loops, whorls, and arches of fingerprints. He analyzes up to a hundred prints an hour—three times the quota.

Microsoft



I tell the man driving the black, smoked glass SUV that I haven't had a minder since my days as a reporter in Baghdad before the first gulf war. He smiles, saying nothing. And before my Iraqi minder let me leave the country, I continue, he made me buy an expensive Saddam wristwatch from his brother's clock shop. "I hope it's cheaper getting out of here than Baghdad," I say.

Out the window I spy a couple of men in dark SWAT uniforms, bouncing across a field on ATVs. "Security," says my minder, whose name is Steve Fischer and who prefers the term "escort." As he wheels the big vehicle along the winding macadam, he explains that the grounds have a nine-mile perimeter and are patrolled by a bunch of these guys.

"How many are there?" I ask. "Can't tell you." Fischer smiles. We've already passed through a guardhouse, where men in similar uniforms peered seriously at ID cards before waving us through. "And what's that?" I ask, pointing toward a satellite dish as big as a bus. "Can't tell you," he says.

"Iraq," I say, "was easier."

I've just entered one of America's most sensitive outposts, the headquarters of the FBI's Criminal Justice Information Services Division (CJIS), in the low mountains of northern West Virginia. It's not missile silos or gold ingots that make CJIS (pronounced SEE-jus)

Waiting to pounce on a virtual criminal, FBI Lt. Julian Galford (above) demonstrates a firearms training simulator to local policemen. At the on-site day-care center, a teacher "marches" children to recess (right).


26306
POPULATION: 0

(26306 is the 986-acre FBI complex.)

CRIME RATE:

No one's talking

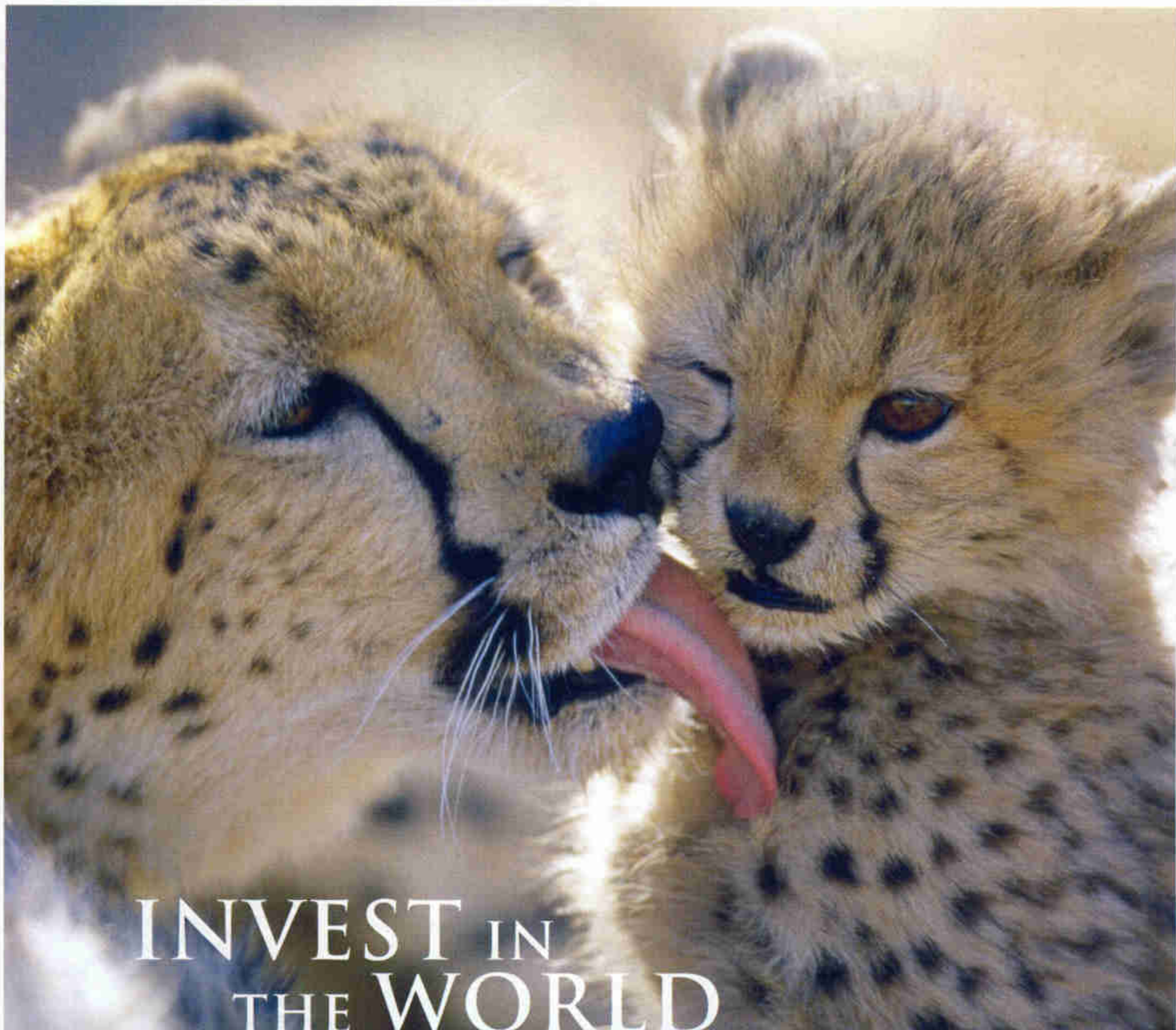
NUMBER OF FINGERPRINTS IN FBI FILES IN 1924:
810,000

TODAY: 252 million

FREQUENCY OF VIOLENT CRIMES IN THE U.S.:

One every 23 seconds





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CLARKSBURG, WEST VIRGINIA

secretive, but information. Hidden away in these gently rolling hills, barely a half mile from a busy interstate highway, is the world's most sophisticated depository of crime data, as well as the largest repository of fingerprints anywhere on the planet.

Ten miles northeast of the town of Clarksburg (birthplace of Stonewall Jackson) and 250 miles from Washington, D.C., it's here, thanks to the powerful West Virginia Senator Robert Byrd, that the FBI put its National Crime Information Center, its Brady Act gun check headquarters, its Uniform Crime Reports data center, its Integrated Automated Fingerprint Identification System, as well as super-sophisticated computers to keep it all humming.

"If Osama bin Laden tries to buy a gun at Wal-Mart, we'll know about it," says Fischer.

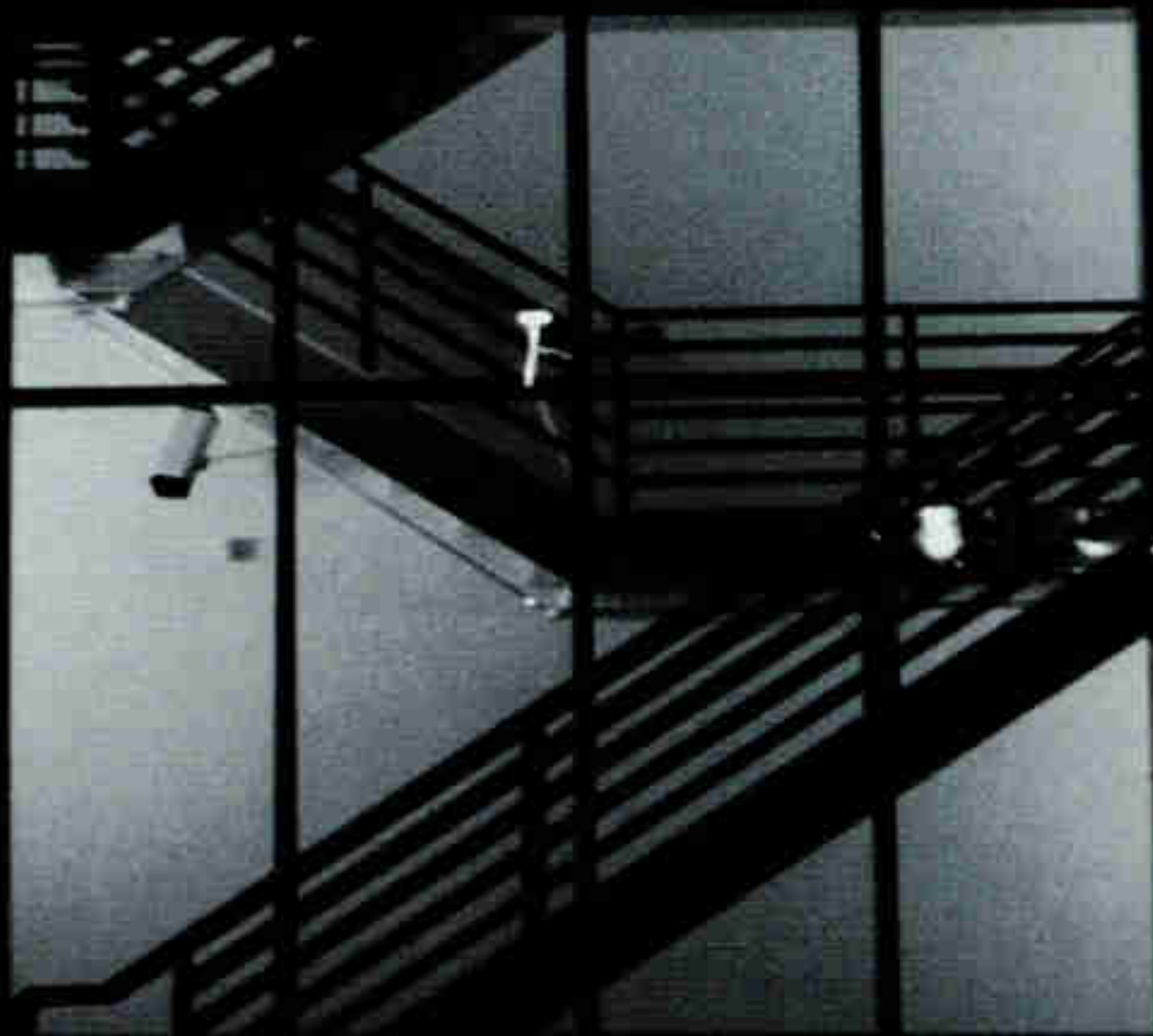
Most of the CJIS operations, including its nearly 3,000 employees, are in a sprawling, three-story building that rises, sphinxlike, on a landscaped hillside in the middle of a forest. Everyone who works here has a top secret clearance. There's no sign on the interstate that announces the complex, only a cryptic notice that Exit 124 will take you to Jerry Dove Drive (named after an FBI agent, a native West Virginian). Such caution is just the way the FBI wants it. And it's why I was never out of Steve Fischer's sights.

No one lives at CJIS, but it's a 24-7 operation with a day-care center, a 600-seat cafeteria, a bank, and a fitness center—although many employees make the whole place a fitness center, using their off time to engage in brisk, determined walks along miles of hallways under soaring atriums. It seems like an ordinary office park until I visit the post office, where mail handlers wearing plastic gloves sit under biohazard hoods in a sealed room. Or until I notice a plaque with the pictures of 34 "FBI Service Martyrs" killed in action—including Jerry Dove himself, slain in one of the bloodiest shoot-outs in the bureau's history, a Miami bank robbery arrest that went awry in 1986.

In the visitors center I hear the pop-pop-pop of gunfire, but it's only a simulated Firearms Training System demonstration for some local police—and as close to real crime as this center gets. (I take "the test," firing my Glock .22 at a screen where a suspected bank robber is going for a rifle and, after scoring three "disabling" shots, am pronounced "very trainable" by FBI Lt. Julian Galford.)

The real crime work here is done amid cubicles and in front of computer screens. "My job is to arrest the numbers and torture them until they confess," Sam Berhanu, an econometrician who runs the Crime Analysis Research and Development Unit, says with a laugh. "No Miranda warnings." Berhanu's unit helps produce the thick annual report, "Crime in the United States," that makes headlines every year as local crime agencies crow about (or hide from) their crime-fighting numbers.

Upstairs, Berhanu's boss, Michael Kirkpatrick (since retired), talks more



By night Paula Clelland trains to secure her third Toughman women's boxing title. By day she tracks health-care fraud with the Financial Crimes Intelligence Unit. "My job," Clelland says, "has given me a lot of confidence as a mother, a wife—and athlete."

MORE THAN JUST A PRETTY INTERFACE.



Sure our new Web site looks great. But, in this case, beauty is more than skin-deep. That's because the new usps.com is filled with easy-to-use tools to help you with just about all your shipping needs. You can pay postage, print labels, add Insurance and order a next-day Carrier Pickup at no charge. Use Priority Mail® service and you also get Delivery Confirmation™ for free. The simple yet powerful usps.com – it's how the U.S. Postal Service® is working for you.



CLARKSBURG, WEST VIRGINIA



like an accountant (which he is) than a crime buster (which he used to be). He describes CJIS's "core line of business," fingerprints, and "our customers," 700,000 cops. Those customers use CJIS's computers to get instant background information about suspects. And according to the daily "success stories" circulated via e-mail to nearly 400 fingerprint examiners, they are happy customers.

The fingerprint unit processes some 50,000 prints every day. With the help of computers that hold around 80 terabytes of information (a single terabyte is the equivalent of a shelf of books about 20 miles long), in minutes prints sent from the field are compared with those in its digital archives. And while most of the "hits" are for garden-variety criminals, the unit helped make the case against Oklahoma City bomber Timothy McVeigh and the Washington, D.C., sniper suspects.

"Each of these little babies can process about 6,000 transactions per second," says Joe Mazzie, Data Center operations manager, pointing to a row of computers that look like double-wide refrigerators. "And we have 12." Mazzie, a local who traces his ancestry to Stonewall Jackson, has a head full of interesting numbers. Some of the busiest days for the gun-buying system, he notes, are Valentine's Day and Mother's Day. "Some people have very different ideas about what a romantic present is," he says with a wry smile.

As we leave, Steve Fischer motions down a hallway. "You ready to go to the gift shop?" he asks. "There's a sale on J. Edgar Hoover watches." □

As one shift leaves, the next begins (above), keeping the FBI humming 24 hours a day. Yet the center's 986 acres—patched together from a reclaimed strip mine, rolling cow pastures, and an old graveyard (below)—still hold remnants of a slower time.



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Use your head. Ordinary phones send sound by air conduction. The small speaker in the earpiece vibrates and sends sound waves through the air. They're received by your ear and interpreted as speech by your brain.

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*This technology is not available in stores...
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How the Miracle Phone™ works

The Miracle Phone's patented DirectVibe™ Pulsator transduces sound waves via bone conduction, bypassing the conventional auditory process. By placing the DirectVibe Pulsator at strategic locations on your head, sound vibrations travel from the pulsator to the speech recognition center in your brain.

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- | | |
|----------------------------|---------------------------|
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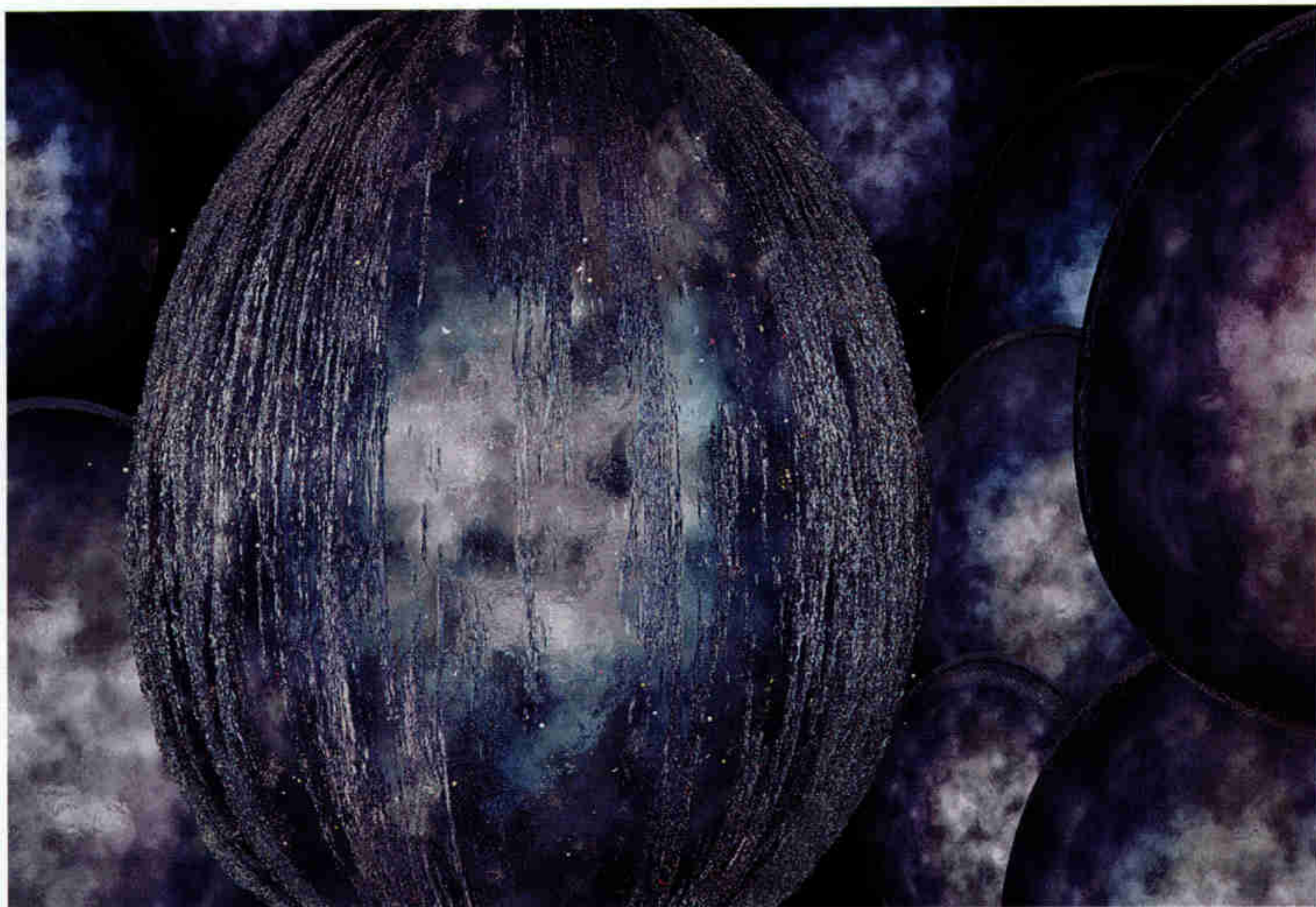
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Final Edit



BEYOND THE BIG BANG

Far-out Visions

How's this for a brainteaser: Draw a patch of infinite space, and populate it with multiple universes. Associate art director Jeff Osborn posed that challenge to artists for a story on the latest thinking in cosmology. Never mind that the multiverse resides in a realm of unproved theoretical physics. "Find a compelling visual metaphor," Osborn instructed. Accepting the dare, Kenneth A. Huff created a sketch of the multiverse as a cluster of ovoids full of star matter (above). Malcolm Godwin of Moonrunner Design imagined universes bubbling off a cosmic force field in many shapes, implying different dimensions and physics. Huff's art entranced the editors with its beauty, but in the end they favored Godwin's vision (pages 110-121), finding it more accessible. "This is weird stuff," Osborn said, "and both artists were willing to get in there and struggle with it."

CUT IT OR KEEP IT? Send the image as an e-greeting and see a Final Edit runner-up at nationalgeographic.com/magazine/0505.

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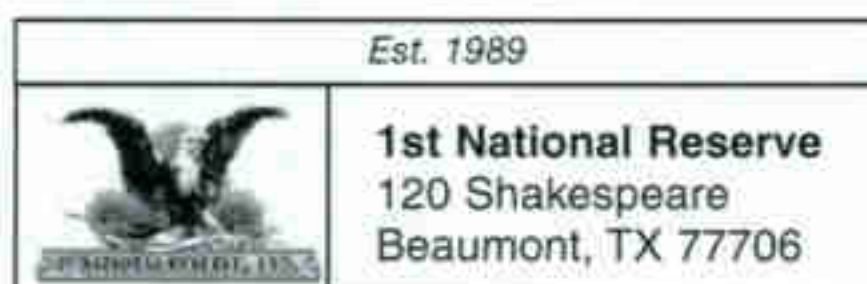
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Do It Yourself

POISON (SEE PAGE 2)



BETTMANN/CORBIS (ABOVE); HULTON ARCHIVE/GETTY IMAGES (RIGHT)

DON'T TRY IT AT HOME

Poisons in Your Pantry

It may be scrubbed spotless, but this 1950s kitchen—and yours—could be hiding poisonous killers. Common products, from air fresheners to insect sprays, contain toxic chemicals. Below are some evils that may lurk in your home. For more, go to householdproducts.nlm.nih.gov.

Cleaning products

Rust removers and drain, toilet-bowl, and oven cleaners can cause chemical burns. Read label directions about wearing gloves and opening windows when using these products.

Bleach It forms a toxic gas if mixed with ammonia or an acidic toilet-bowl cleaner.

Lead Leached from old lead pipes, it can contaminate drinking water and damage the central nervous system, especially in children.

Air pollutants The air in some homes can be more polluted than the outside air in an industrialized city. Make sure air-supply vents are unblocked, and test for radon, a naturally occurring gas that, at unhealthy levels, can cause lung cancer.

Windshield wiper fluid Easily absorbed by inhaling or skin contact, it can damage organs and the nervous system. Wear gloves when adding fluid and do so in a well-ventilated place.

Vitamins and minerals

Even common supplements can bother some people; iron pills particularly can be toxic.

Pet Peeves

Antifreeze Toxic to pets, so watch for fluid leaked from cars onto garage floors and driveways.

Rodent bait Keep traps out of reach of pets; eating poisonous bait prevents blood from clotting and can be fatal.

Plants Ingesting such common plants as ivies, azaleas, and daffodils damages a pet's digestive system and can also be fatal.

Foods Many pets can't tolerate chocolate, coffee, avocados, raisins, grapes, onions, garlic, yeast dough—even salt.

PICKS

3 characters

Staff writer **Cathy**

Newman, author of the poison story, has her own favorites among history's, and fiction's, poisoned personalities.

■ **Rasputin** The Russian mystic (below) wielded so much influence on the tsar's family that in 1916 a group of aristocrats had his food laced with poison—to no avail. They had to shoot then drown him to get the job done.



■ Poison Ivy

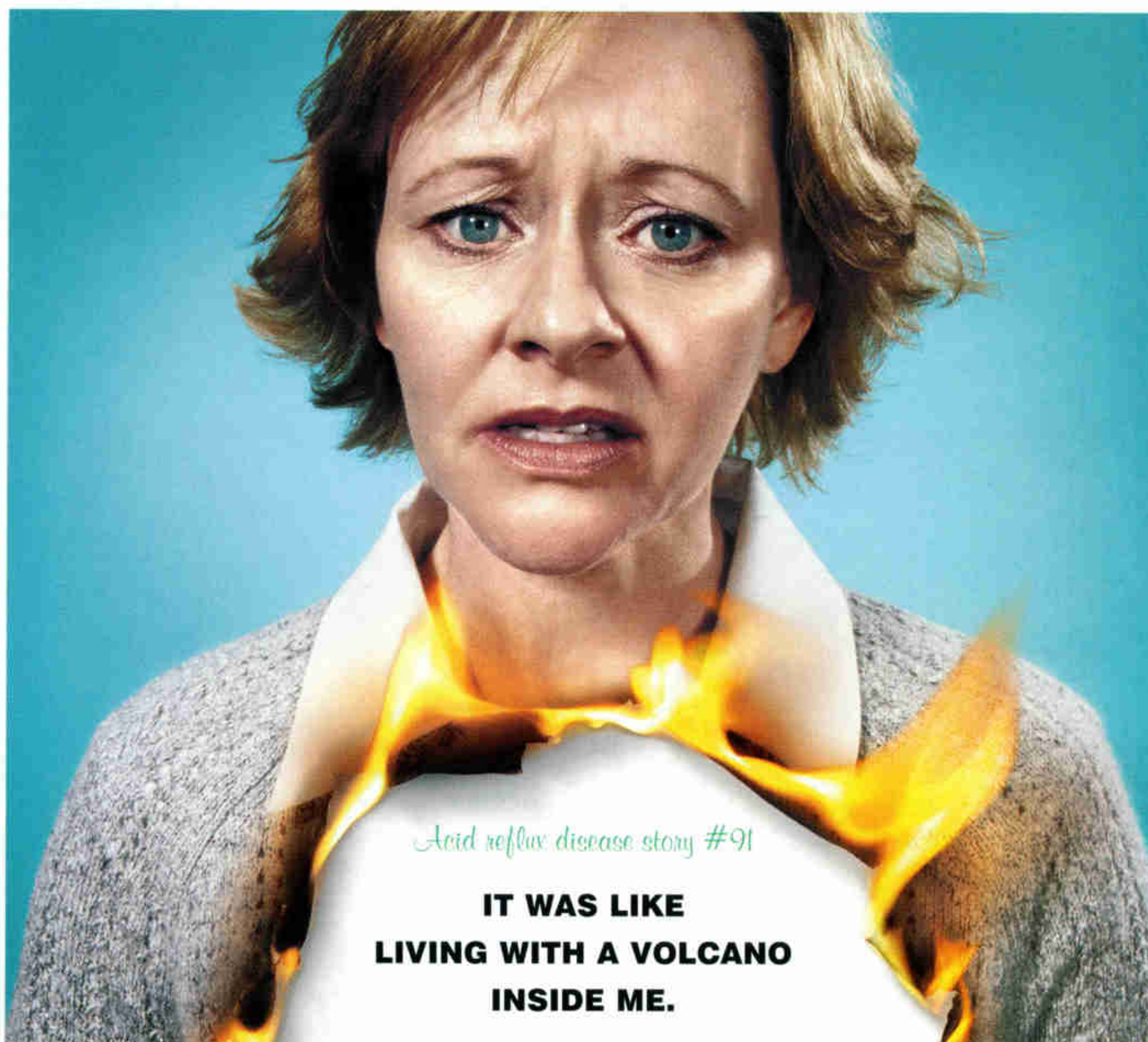
The fictional creator of toxins in Gotham—and Batman's nemesis—often relied on poisoned perfumes and lipsticks to subdue her victims.

■ Ludwig van Beethoven

A lock of the composer's hair tested positive for lead poisoning, which may explain why Beethoven suffered from chronic health problems.

POISONOUS PICS

Check out our lethal online photo gallery and video at nationalgeographic.com/magazine/0505.



Acid reflux disease story #91

**IT WAS LIKE
LIVING WITH A VOLCANO
INSIDE ME.**

People can have different experiences with acid reflux disease. Some get a bitter taste in their mouth, some feel severe heartburn pain after a meal, while others can even develop a chronic cough. Fortunately, there's Prevacid®. It can be taken in many forms — one that's a pill, one you can drink, and one that disintegrates in your mouth — to help treat heartburn and many other kinds of symptoms related to acid reflux disease in many kinds of people. Maybe it's time to see if it can help yours. So ask your doctor if there's a Prevacid that's right for you.



Put it to your acid test.

For a **FREE trial certificate**, call **1-866-5PREVACID** or visit **prevacid.com** today.

If you suffer from persistent heartburn two or more days a week, despite treatment and diet changes, it may be acid reflux disease (ARD). Heartburn is the most common symptom of ARD. Prevacid Capsules, Prevacid for Oral Suspension, and Prevacid® SoluTab™ (lansoprazole) Orally Disintegrating Tablets are used to treat ARD. Individual results may vary. Prescription Prevacid has a low occurrence of side effects such as diarrhea, abdominal pain, and nausea. Symptom relief does not rule out serious stomach conditions. Please see adjacent brief summary of important information and talk to your doctor.

Brief Summary of Prescribing Information
(Nos. 1541, 1543, 1544, 3046, 7309, 7311)
03-5366-R24-Brt. Rev. July, 2004

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PREVACID® (lansoprazole) For Delayed-Release Oral Suspension

PREVACID® SoluTab™ (lansoprazole)

Delayed-Release Orally Disintegrating Tablets Rx only

PREVACID Delayed-Release Capsules, PREVACID SoluTab Delayed-Release Orally Disintegrating Tablets and PREVACID For Delayed-Release Oral Suspension are indicated for:

Short-Term Treatment (4 weeks) of Active Duodenal Ulcer

H. pylori Eradication to Reduce the Risk of Duodenal Ulcer Recurrence

Triple Therapy: PREVACID/amoxicillin/clarithromycin

Dual Therapy: PREVACID/amoxicillin

Who are either allergic or intolerant to clarithromycin or in whom resistance to clarithromycin is known or suspected.

Maintenance of Healed Duodenal Ulcers

Controlled studies do not extend beyond 12 months.

Short-Term Treatment (up to 8 weeks) of Active Benign Gastric Ulcer

Healing of NSAID-Associated Gastric Ulcer

In patients who continue NSAID use. Controlled studies did not extend beyond 8 weeks.

Risk Reduction of NSAID-Associated Gastric Ulcer

In patients with a history of a documented gastric ulcer who require the use of an NSAID. Controlled studies did not extend beyond 12 weeks

Gastroesophageal Reflux Disease (GERD)

Short-Term Treatment of Symptomatic GERD

Short-Term Treatment (up to 8 weeks) of Erosive Esophagitis

For patients who do not heal with PREVACID for 8 weeks (5-10%), it may be helpful to give an additional 8 weeks of treatment. If there is a recurrence of erosive esophagitis an additional 8-week course of PREVACID may be considered.

Maintenance of Healing of Erosive Esophagitis

Controlled studies did not extend beyond 12 months.

Pathological Hypersecretory Conditions including Zollinger-Ellison Syndrome

CONTRAINDICATIONS

PREVACID is contraindicated in patients with known hypersensitivity to any component of the formulation of PREVACID.

Amoxicillin is contraindicated in patients with a known hypersensitivity to any penicillin.

Clarithromycin is contraindicated in patients with a known hypersensitivity to clarithromycin, erythromycin, and any of the macrolide antibiotics.

Concomitant administration of clarithromycin with cisapride, pimozide, astemizole, or terfenadine is contraindicated. There have been post-marketing reports of drug interactions when clarithromycin and/or erythromycin are co-administered with cisapride, pimozide, astemizole, or terfenadine resulting in cardiac arrhythmias (QT prolongation, ventricular tachycardia, ventricular fibrillation, and torsades de pointes) most likely due to inhibition of metabolism of these drugs by erythromycin and clarithromycin. Fatalities have been reported.

(Please refer to full prescribing information for amoxicillin and clarithromycin before prescribing.)

WARNINGS

CLARITHROMYCIN SHOULD NOT BE USED IN PREGNANT WOMEN EXCEPT IN CLINICAL CIRCUMSTANCES WHERE NO ALTERNATIVE THERAPY IS APPROPRIATE. IF PREGNANCY OCCURS WHILE TAKING CLARITHROMYCIN, THE PATIENT SHOULD BE APPRISED OF THE POTENTIAL HAZARD TO THE FETUS. (SEE WARNINGS IN PRESCRIBING INFORMATION FOR CLARITHROMYCIN.)

Pseudomembranous colitis has been reported with nearly all antibacterial agents, including clarithromycin and amoxicillin, and may range in severity from mild to life threatening. Therefore, it is important to consider this diagnosis in patients who present with diarrhea subsequent to the administration of antibacterial agents.

Treatment with antibacterial agents alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is a primary cause of "antibiotic-associated colitis."

After the diagnosis of pseudomembranous colitis has been established, therapeutic measures should be initiated. Mild cases of pseudomembranous colitis usually respond to discontinuation of the drug alone. In moderate to severe cases, consideration should be given to management with fluids and electrolytes, protein supplementation, and treatment with an antibacterial drug clinically effective against *Clostridium difficile* colitis.

Serious and occasionally fatal hypersensitivity (anaphylactic) reactions have been reported in patients on penicillin therapy. These reactions are more apt to occur in individuals with a history of penicillin hypersensitivity and/or a history of sensitivity to multiple allergens.

There have been well-documented reports of individuals with a history of penicillin hypersensitivity reactions who have experienced severe hypersensitivity reactions when treated with a cephalosporin. Before initiating therapy with any penicillin, careful inquiry should be made concerning previous hypersensitivity reactions to penicillins, cephalosporins, and other allergens. If an allergic reaction occurs, amoxicillin should be discontinued and the appropriate therapy instituted.

SERIOUS ANAPHYLACTIC REACTIONS REQUIRE IMMEDIATE EMERGENCY TREATMENT WITH EPINEPHRINE, OXYGEN, INTRAVENOUS STEROIDS, AND AIRWAY MANAGEMENT, INCLUDING INTUBATION, SHOULD ALSO BE ADMINISTERED AS INDICATED.

PRECAUTIONS

General

Symptomatic response to therapy with lansoprazole does not preclude the presence of gastric malignancy.

Information for Patients

PREVACID is available as a capsule, orally disintegrating tablet and oral suspension, and is available in 15 mg and 30 mg strengths. Directions for use specific to the route and available methods of administration for each of these dosage forms is presented below. PREVACID should be taken before eating. PREVACID products SHOULD NOT BE CRUSHED OR CHEWED.

Phenylethanolorics: Contains Phenylalanine 2.5 mg per 15 mg Tablet and 5.1 mg per 30 mg Tablet.

Administration Options

1. PREVACID Delayed-Release Capsules

PREVACID Delayed-Release Capsules should be swallowed whole.

Alternatively, for patients who have difficulty swallowing capsules, PREVACID Delayed-Release Capsules can be opened and administered as follows:

- Open capsule.
- Sprinkle intact granules on one tablespoon of either applesauce, ENSURE® pudding, cottage cheese, yogurt or strained pears.
- Swallow immediately.

PREVACID Delayed-Release Capsules may also be emptied into a small volume of either apple juice, orange juice or tomato juice and administered as follows:

- Open capsule.
- Sprinkle intact granules into a small volume of either apple juice, orange juice or tomato juice (60 mL – approximately 2 ounces).
- Mix briefly.
- Swallow immediately.
- To ensure complete delivery of the dose, the glass should be rinsed with two or more volumes of juice and the contents swallowed immediately.

USE IN OTHER FOODS AND LIQUIDS HAS NOT BEEN STUDIED CLINICALLY AND IS THEREFORE NOT RECOMMENDED.

2. PREVACID SoluTab Delayed-Release Orally Disintegrating Tablets

PREVACID SoluTab should not be chewed. Place the tablet on the tongue and allow it to disintegrate, with or without water, until the particles can be swallowed. The tablet typically disintegrates in less than 1 minute.

Alternatively, for children or other patients who have difficulty swallowing tablets, PREVACID SoluTab can be delivered in two different ways.

PREVACID SoluTab – Oral Syringe

For administration via oral syringe, PREVACID SoluTab can be administered as follows:

- Place a 15 mg tablet in oral syringe and draw up approximately 4 mL of water, or place a 30 mg tablet in oral syringe and draw up approximately 10 mL of water.
- Shake gently to allow for a quick dispersal.
- After the tablet has dispersed, administer the contents within 15 minutes.
- Refill the syringe with approximately 2 mL (5 mL for the 30 mg tablet) of water, shake gently, and administer any remaining contents.

PREVACID SoluTab – Nasogastric Tube Administration (≥ 6 French)

For administration via a nasogastric tube, PREVACID SoluTab can be administered as follows:

- Place a 15 mg tablet in a syringe and draw up 4 mL of water, or place a 30 mg tablet in a syringe and draw up 10 mL of water.
- Shake gently to allow for a quick dispersal.
- After the tablet has dispersed, inject through the nasogastric tube into the stomach within 15 minutes.
- Refill the syringe with approximately 5 mL of water, shake gently, and flush the nasogastric tube.

3. PREVACID for Delayed-Release Oral Suspension

PREVACID for Delayed-Release Oral Suspension should be administered as follows:

- Open packet.
- To prepare a dose, empty the packet contents into a container containing 2 tablespoons of WATER. DO NOT USE OTHER LIQUIDS OR FOODS.
- Stir well, and drink immediately.
- If any material remains after drinking, add more water, stir, and drink immediately.
- **This product should not be given through enteral administration tubes.**

Drug Interactions

Lansoprazole is metabolized through the cytochrome P₄₅₀ system, specifically through the CYP3A and CYP2C19 isozymes. Studies have shown that lansoprazole does not have clinically significant interactions with other drugs metabolized by the cytochrome P₄₅₀ system, such as warfarin, antipyrine, indomethacin, ibuprofen, phenytoin, propranolol, prednisone, diazepam, or clarithromycin in healthy subjects. These compounds are metabolized through various cytochrome P₄₅₀ isozymes including CYP1A2, CYP2C8, CYP2C19, CYP2D6, and CYP3A. When lansoprazole was administered concomitantly with theophylline (CYP1A2, CYP3A), a minor increase (10%) in the clearance of theophylline was seen. Because of the small magnitude and the direction of the effect on theophylline clearance, this interaction is unlikely to be of clinical concern. Nonetheless, individual patients may require additional titration of their theophylline dosage when lansoprazole is started or stopped to ensure clinically effective blood levels.

In a study of healthy subjects neither the pharmacokinetics of warfarin enantiomers nor prothrombin time were affected following single or multiple 60 mg doses of lansoprazole. However, there have been reports of increased International Normalized Ratio (INR) and prothrombin time in patients receiving proton pump inhibitors, including lansoprazole, and warfarin concomitantly. Increases in INR and prothrombin time may lead to abnormal bleeding and even death. Patients treated with proton pump inhibitors and warfarin concomitantly may need to be monitored for increases in INR and prothrombin time.

Lansoprazole has also been shown to have no clinically significant interaction with amoxicillin.

In a single-dose crossover study examining lansoprazole 30 mg and omeprazole 20 mg each administered alone and concomitantly with sucralfate 1 gram, absorption of the proton pump inhibitors was delayed and their bioavailability was reduced by 17% and 16%, respectively, when administered concomitantly with sucralfate. Therefore, proton pump inhibitors should be taken at least 30 minutes prior to sucralfate. In clinical trials, antacids were administered concomitantly with PREVACID Delayed-Release Capsules; this did not interfere with its effect.

Lansoprazole causes a profound and long-lasting inhibition of gastric acid secretion; therefore, it is theoretically possible that lansoprazole may interfere with the absorption of drugs where gastric pH is an important determinant of bioavailability (e.g., ketoconazole, ampicillin esters, iron salts, digoxin).

Carcinogenesis, Mutagenesis, Impairment of Fertility

In two 24-month carcinogenicity studies, Sprague-Dawley rats were treated orally with doses of 5 to 150 mg/kg/day, about 1 to 40 times the exposure on a body surface (mg/m²) basis, of a 50-kg person of average height (1.46 m² body surface area) given the recommended human dose of 30 mg/day (22.2 mg/m²). Lansoprazole produced dose-related gastric enterochromaffin-like (ECL) cell hyperplasia and ECL cell carcinoids in both male and female rats. It also increased the incidence of intestinal metaplasia of the gastric epithelium in both sexes. In male rats, lansoprazole produced a dose-related increase of testicular interstitial cell adenomas. The incidence of these adenomas in rats receiving doses of 15 to 150 mg/kg/day (4 to 40 times the recommended human dose based on body surface area) exceeded the low background incidence (range = 1.4 to 10%) for this strain of rat. Testicular interstitial cell adenoma also occurred in 1 of 30 rats treated with 50 mg/kg/day (13 times the recommended human dose based on body surface area) in a 1-year toxicity study.

In a 24-month carcinogenicity study, CD-1 mice were treated orally with doses of 15 to 600 mg/kg/day, 2 to 80 times the recommended human dose based on body surface area. Lansoprazole produced a dose-related increased incidence of gastric ECL cell hyperplasia. It also produced an increased incidence of liver tumors (hepatocellular adenoma plus carcinoma). The tumor incidences in male mice treated with 300 and 600 mg/kg/day (40 to 80 times the recommended human dose based on body surface area) and female mice treated with 150 to 600 mg/kg/day (20 to 80 times the recommended human dose based on body surface area) exceeded the ranges of background incidences in historical controls for this strain of mice. Lansoprazole treatment produced adenoma of rete testis in male mice receiving 75 to 600 mg/kg/day (10 to 80 times the recommended human dose based on body surface area).

Lansoprazole was not genotoxic in the Ames test, the *ex vivo* rat hepatocyte unscheduled DNA synthesis (UDS) test, the *in vivo* mouse micronucleus test or the rat bone marrow cell chromosomal aberration test. It was positive in *in vitro* human lymphocyte chromosomal aberration assays.

Lansoprazole at oral doses up to 150 mg/kg/day (40 times the recommended human dose based on body surface area) was found to have no effect on fertility and reproductive performance of male and female rats.

Pregnancy: Teratogenic Effects.

Pregnancy Category B

Lansoprazole

Teratology studies have been performed in pregnant rats at oral doses up to 150 mg/kg/day (40 times the recommended human dose based on body surface area) and pregnant rabbits at oral doses up to 30 mg/kg/day (16 times the recommended human dose based on body surface area) and have revealed no evidence of impaired fertility or harm to the fetus due to lansoprazole.

There are, however, no adequate or well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Pregnancy Category C

Clarithromycin

See WARNINGS (above) and full prescribing information for clarithromycin before using in pregnant women.

Nursing Mothers

Lansoprazole or its metabolites are excreted in the milk of rats. It is not known whether lansoprazole is excreted in human milk. Because many drugs are excreted in human milk, because of the potential for serious adverse reactions in nursing infants from lansoprazole, and because of the potential for tumorigenicity shown for lansoprazole in rat carcinogenicity studies, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use

The safety and effectiveness of PREVACID have been established in pediatric patients 1 to 17 years of age for short-term treatment of symptomatic GERD and erosive esophagitis. Use of PREVACID in this population is supported by evidence from adequate and well-controlled studies of PREVACID in adults with additional clinical, pharmacokinetic, and pharmacodynamic studies performed in pediatric patients. The adverse events profile in pediatric patients is similar to that of adults. There were no adverse events reported in U.S. clinical studies that were not previously observed in adults. The safety and effectiveness of PREVACID in patients <1 year of age have not been established.

1 to 11 years of age

The pediatric safety of PREVACID Delayed-Release Capsules has been assessed in 66 pediatric patients aged 1 to 11 years of age. Of the 66 patients with GERD 85% (56/66) took PREVACID for 8 weeks and 15% (10/66) took it for 12 weeks.

The most frequently reported (2 or more patients) treatment-related adverse events in patients 1 to 11 years of age (N=66) were constipation (5%) and headache (3%).

12 to 17 years of age

The safety of PREVACID Delayed-Release Capsules has been assessed in these 87 adolescent patients. Of the 87 adolescent patients with GERD, 6% (5/87) took PREVACID for <6 weeks, 93% (81/87) for 6-10 weeks, and 1% (1/87) for >10 weeks.

The most frequently reported (at least 3%) treatment-related adverse events in these patients were headache (7%), abdominal pain (5%), nausea (3%) and dizziness (3%). Treatment-related dizziness, reported in this package insert as occurring in <1% of adult patients, was reported in this study by 3 adolescent patients with nonerosive GERD, who had dizziness concurrently with other events (such as migraine, dyspnea, and vomiting).

Use in Women

Over 4,000 women were treated with lansoprazole. Ulcer healing rates in females were similar to those in males. The incidence rates of adverse events were also similar to those seen in males.

Use in Geriatric Patients

Ulcer healing rates in elderly patients are similar to those in a younger age group. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in younger patients. For elderly patients, dosage and administration of lansoprazole need not be altered for a particular indication.

ADVERSE REACTIONS

Clinical

Worldwide, over 10,000 patients have been treated with lansoprazole in Phase 2-3 clinical trials involving various dosages and durations of treatment. The adverse reaction profiles for PREVACID Delayed-Release Capsules and PREVACID for Delayed-Release Oral Suspension are similar. In general, lansoprazole treatment has been well-tolerated in both short-term and long-term trials.

The following adverse events were reported by the treating physician to have a possible or probable relationship to drug in 1% or more of PREVACID-treated patients and occurred at a greater rate in PREVACID-treated patients than placebo-treated patients:

Incidence of Possibly or Probably Treatment-Related Adverse Events in Short-Term, Placebo-Controlled Studies		
	PREVACID (N= 2768)	Placebo (N= 1023)
Body System/Adverse Event	%	%
Body as a Whole		
Abdominal Pain	2.1	1.2
Digestive System		
Constipation	1.0	0.4
Diarrhea	3.8	2.3
Nausea	1.3	1.2

Headache was also seen at greater than 1% incidence but was more common on placebo. The incidence of diarrhea was similar between patients who received placebo and patients who received lansoprazole 15 mg and 30 mg, but higher in the patients who received lansoprazole 60 mg (2.9%, 1.4%, 4.2%, and 7.4%, respectively).

The most commonly reported possibly or probably treatment-related adverse event during maintenance therapy was diarrhea.

In the risk reduction study of PREVACID for NSAID-associated gastric ulcers, the incidence of diarrhea for patients treated with PREVACID was 5%, misoprostol 22%, and placebo 3%. Additional adverse experiences occurring in <1% of patients or subjects in domestic trials are shown below. Refer to **Postmarketing** for adverse reactions occurring since the drug was marketed.

Body as a Whole – abdomen enlarged, allergic reaction, asthenia, back pain, candidiasis, carcinoma, chest pain (not otherwise specified), chills, edema, fever, flu syndrome, halitosis, infection (not otherwise specified), malaise, neck pain, neck rigidity, pain, pelvic pain. *Cardiovascular System* – angina, arrhythmia, bradycardia, cerebrovascular accident/cerebral infarction, hypertension/hypotension, migraine, myocardial infarction, palpitations, shock (circulatory failure), syncope, tachycardia, vasodilation; *Digestive System* – abnormal stools, anorexia, bezoar, cardiospasm, cholelithiasis, colitis, dry mouth, dyspepsia, dysphagia, enteritis, eructation, esophageal stenosis, esophageal ulcer, esophagitis, fecal discoloration, flatulence, gastric nodules/fundic gland polyps, gastritis, gastroenteritis, gastrointestinal anomaly, gastrointestinal disorder, gastrointestinal hemorrhage, glossitis, gum hemorrhage, hematemesis, increased appetite, increased salivation, melena, mouth ulceration, nausea and vomiting, nausea and vomiting and diarrhea, oral moniliasis, rectal disorder, rectal hemorrhage, stomatitis, tenesmus, thirst, tongue disorder, ulcerative colitis, ulcerative stomatitis; *Endocrine System* – diabetes mellitus, goiter, hypothyroidism; *Hemic and Lymphatic System* – anemia, hemolysis, lymphadenopathy; *Metabolic and Nutritional Disorders* – gout, dehydration, hyperglycemia/hypoglycemia, peripheral edema, weight gain/loss; *Musculoskeletal System* – arthralgia, arthritis, bone disorder, joint disorder, leg cramps, musculoskeletal pain, myalgia, myasthenia, synovitis; *Nervous System* – abnormal dreams, agitation, amnesia, anxiety, apathy, confusion, convulsion, depersonalization, depression, diplopia, dizziness, emotional lability, hallucinations, hemiplegia, hostility aggravated, hyperkinesia, hypertonia, hypesthesia, insomnia, libido decreased/increased, nervousness, neurosis, paresthesia, sleep disorder, somnolence, thinking abnormality, tremor, vertigo; *Respiratory System* – asthma, bronchitis, cough increased, dyspnea, epistaxis, hemoptysis, hiccup, laryngeal neoplasia, pharyngitis, pleural disorder, pneumonia, respiratory disorder, upper respiratory inflammation/infection, rhinitis, sinusitis, stridor; *Skin and Appendages* – acne, alopecia, contact dermatitis, dry skin, fixed eruption, hair disorder, maculopapular rash, nail disorder, pruritus, rash, skin carcinoma, skin disorder, sweating, urticaria; *Special Senses* – abnormal vision, blurred vision, conjunctivitis, deafness, dry eyes, ear disorder, eye pain, otitis media, parosmia, photophobia, retinal degeneration, taste loss, taste perversion, tinnitus, visual field defect; *Urogenital System* – abnormal menses, breast enlargement, breast pain, breast tenderness, dysmenorrhea, dysuria, gynecostasia, impotence, kidney calculus, kidney pain, leukorrhea, menorrhagia, menstrual disorder, penis disorder, polyuria, testis disorder, urethral pain, urinary frequency, urinary tract infection, urinary urgency, urination impaired, vaginitis.

Postmarketing

On-going Safety Surveillance: Additional adverse experiences have been reported since lansoprazole has been marketed. The majority of these cases are foreign-sourced and a relationship to lansoprazole has not been established. Because these events were reported voluntarily from a population of unknown size, estimates of frequency cannot be made. These events are listed below by COSTART body system.

Body as a Whole – anaphylactoid-like reaction; *Digestive System* – hepatotoxicity, pancreatitis, vomiting; *Hemic and Lymphatic System* – agranulocytosis, aplastic anemia, hemolytic anemia, leukopenia, neutropenia, pancytopenia, thrombocytopenia, and thrombotic thrombocytopenic purpura; *Skin and Appendages* – severe dermatologic reactions including erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis (some fatal); *Special Senses* – speech disorder; *Urogenital System* – urinary retention.

Combination Therapy with Amoxicillin and Clarithromycin

In clinical trials using combination therapy with PREVACID plus amoxicillin and clarithromycin, and PREVACID plus amoxicillin, no adverse reactions peculiar to these drug combinations were observed. Adverse reactions that have occurred have been limited to those that had been previously reported with PREVACID, amoxicillin, or clarithromycin.

Triple Therapy: PREVACID/amoxicillin/clarithromycin

The most frequently reported adverse events for patients who received triple therapy for 14 days were diarrhea (7%), headache (6%), and taste perversion (5%). There were no statistically significant differences in the frequency of reported adverse events between the 10- and 14-day triple therapy regimens. No treatment-emergent adverse events were observed at significantly higher rates with triple therapy than with any dual therapy regimen.

Dual Therapy: PREVACID/amoxicillin

The most frequently reported adverse events for patients who received PREVACID t.i.d. plus amoxicillin t.i.d. dual therapy were diarrhea (8%) and headache (7%). No treatment-emergent adverse events were observed at significantly higher rates with PREVACID t.i.d. plus amoxicillin t.i.d. dual therapy than with PREVACID alone.

For more information on adverse reactions with amoxicillin or clarithromycin, refer to their package inserts. **ADVERSE REACTIONS** sections.

Laboratory Values

The following changes in laboratory parameters for lansoprazole were reported as adverse events:

Abnormal liver function tests, increased SGOT (AST), increased SGPT (ALT), increased creatinine, increased alkaline phosphatase, increased globulins, increased GGTP, increased/decreased/abnormal WBC, abnormal AG ratio, abnormal RBC, bilirubinemia, eosinophilia, hyperlipemia, increased/decreased electrolytes, increased/decreased cholesterol, increased glucocorticoids, increased LDH, increased/decreased/abnormal platelets, and increased gastrin levels. Urine abnormalities such as albuminuria, glycosuria, and hematuria were also reported. Additional isolated laboratory abnormalities were reported. In the placebo controlled studies, when SGOT (AST) and SGPT (ALT) were evaluated, 0.4% (4/978) placebo patients and 0.4% (11/2677) lansoprazole patients had enzyme elevations greater than three times the upper limit of normal range at the final treatment visit. None of these lansoprazole patients reported jaundice at any time during the study.


In clinical trials using combination therapy with PREVACID plus amoxicillin and clarithromycin, and PREVACID plus amoxicillin, no increased laboratory abnormalities particular to these drug combinations were observed.

For more information on laboratory value changes with amoxicillin or clarithromycin, refer to their package inserts. **ADVERSE REACTIONS** section.

OVERDOSAGE

Oral doses up to 5000 mg/kg in rats (approximately 1300 times the recommended human dose based on body surface area) and mice (about 675.7 times the recommended human dose based on body surface area) did not produce deaths or any clinical signs.

Lansoprazole is not removed from the circulation by hemodialysis. In one reported case of overdose, the patient consumed 600 mg of lansoprazole with no adverse reaction.

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Flashback



CORAL REEF COLOR

A Bit Too Flashy

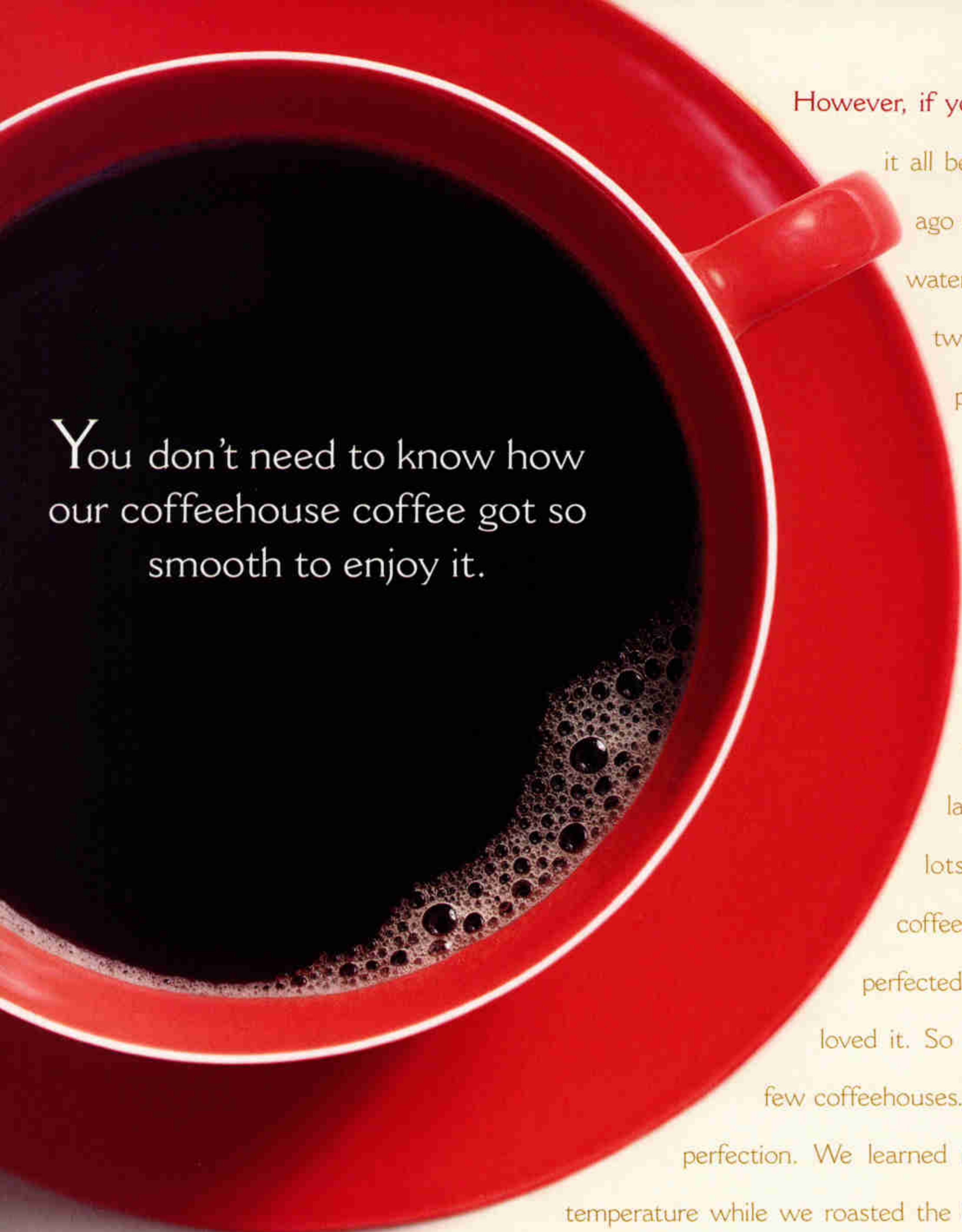
The world's earliest underwater color photography began with a bang. Ichthyologist W. H. Longley and GEOGRAPHIC photographer Charles Martin spent months coordinating divers and dory boats off Florida's Dry Tortugas to take the first ever underwater Autochromes of sea life (including the hogfish, above) for the January 1927 issue. To light the submarine world, pontoon-borne pans of magnesium powder explosives (right) were "discharged by the submerged photographer at the exact moment of his finny subjects' best posings."

The resulting "blinding and deafening detonation" had drawbacks. "On one occasion," the article notes, "Dr. Longley was seriously burned and incapacitated for six days by a premature explosion of an ounce of powder." —Margaret G. Zackowitz



CHARLES MARTIN AND W. H. LONGLEY (TOP); CHARLES MARTIN

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our coffeehouse coffee got so
smooth to enjoy it.

However, if you're curious,
it all began 35 years
ago on the Seattle
waterfront. We had
two things: an old
peanut roaster,
and a desire
to create
a smoother
coffee that
was full of
flavor. It took
late nights, and
lots of cups of
coffee, but we finally
perfected it. And people
loved it. So we opened a
few coffeehouses. And pursued
perfection. We learned to adjust the

temperature while we roasted the beans, so our
coffees are smoother. And we created varieties such as Henry's Blend.[®] Which, incidentally,
is named after the cat that hung around our roastery. And because we were so particular in
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